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An Address.[1]

ON SOME ASPECTS OF CONGRESS.

By W. KEVERALL MCINTYRE, M.C., M.D., B.E.,
Retiring President of the Tasmanian Branch of the British Medical Association.

I DO not propose tonight to read a formal paper, having nothing of an original nature to present for your consideration. It seemed to me that a few reflections on the more outstanding lessons to be learnt from that very important event to us, the recent congress, might not be out of place.

I should like first of all to congratulate the President of the Congress and the Joint Secretaries, Dr. H. N. Butler and Dr. W. L. Crowther, on the very happy outcome of all their labours. One also includes, of course, the various office-bearers, secretaries of sections, and members of committee, both men and women, who gave up so much of their time and worked so unselfishly to make this Tasmanian Congress the undoubted success it was. But I think all of you will agree that the main responsibility rested on the shoulders of those I have named.

When, rather than allow it to lapse, we discussed at a full meeting of the Branch the question of sending an invitation to the Federal Council to hold the congress in Tasmania, some among our members were afraid that with less than half the time usually allowed for preparation and organization, with our hospital difficulties, and with our lack of clinical material, we would be undertaking too great a responsibility. I think that some anxiety over these matters was natural, even though we all realized the importance to the public and ourselves were such a gathering of professional men to take place in Tasmania. Our Joint Secretaries were full of enthusiasm and optimism, and agreed

[1] Read at the annual meeting of the Tasmanian Branch of the British Medical Association on March 24, 1934.

to shoulder the responsibility, if our invitation was sent and accepted; and I have no hesitation in saying that they successfully laid all our doubts to rest and that their optimism was more than justified in the great work that they did.

I thought it a pity that in this, as in other congresses I have attended, more time was not allowed for general discussion after the papers had been read on the subjects set down for full meetings of Congress. Some speakers exceeded their time limit in spite of the President's bell, and this left no time for more than a hasty general discussion. One noticed this both on the "Hospital Problem" and "Cancer Research" days. A suggestion was made, and I think it one well worth considering, that even if some of the sections were crowded up a bit, a whole day might be set aside for the main subject for which full meetings of Congress are held. If the morning could be devoted to the prepared papers and the general discussions could be continued after the luncheon hour, men would have time to think over what had been read, and the value and interest of the meeting would be greatly increased. I feel sure we missed what would have proved a very lively discussion on cancer research had we had, say, two hours in the afternoon for a general discussion.

Hospitals.

It would be difficult to find more interesting and important problems than the two selected for the main discussions of Congress. The hospital question presents peculiar difficulties in that the various States have already progressed along rather divergent paths in their efforts to solve it. The old honorary system, which up to recent years has been rigidly upheld by the British Medical Association, has now become such a tax on the medical profession that a review of the system is inevitable and long overdue. That is generally recognized and need not be discussed. The difficulty lies in finding a common hospital policy that will satisfy the diverse conditions in the various States and be acceptable to our own Branches and the State Governments who subsidize the public hospitals. The attitude of the State Governments towards the hospital problem differs. Some are sympathetic to our point of view and willing to cooperate in an effort to find a solution which is fair to the public and to ourselves, while others are apparently agreeable to go to any lengths in exploiting the profession. Unfortunately we, as a profession, have not yet formulated a common hospital policy. According to Dr. Duhig, Queensland is within measureable distance of complete nationalization. The local Branch seems to have been dragged at the chariot wheels of the Government. Western Australia is travelling the same road and seems to be in much the same position as Tasmania. South Australia, from Dr. Leonard Lindon's paper, seems to be in the happiest position of all the States. Well equipped country hospitals are built and run by the Government on community lines. Intermediate and private patients from whom proportional fees may be collected by practitioners predominate, but charity cases are also accommodated. Adelaide Hospital, like the Melbourne and Sydney large public hospitals, as far as possible admits only those unable to pay private fees. Dr. Lindon did not say whether intermediate hospitals were being built in Adelaide. Victoria, with a sympathetic Government cooperating, is attempting to limit the public hospitals to the really poor, with the present honorary staffing system, and to provide independent intermediate hospitals for those able to pay moderate fees for hospital services and to their doctor; the private hospitals are still to be available to those able to pay on a higher scale. The New South Wales Government in power at present is also in sympathy with the State Branch, which favours the community hospital system, and Dr. Hunter quoted instances in metropolitan hospitals where this system has been introduced on a limited scale.

Dr. Ramsay and Dr. Fay, speaking for Tasmania, pointed out the unfortunate position of the profession in this State under the present *Hospitals Act*. The Act in effect says that since the Government contributes largely towards the upkeep of the public hospitals, all persons, irrespective of their means, have the right to make use of them, and as long as this act is on the statutes no solution fair to ourselves seems possible. There is no doubt that we should have a member of our Association in Parliament, who would have the requisite knowledge to lay our point of view before the House when questions of policy affecting us arose.

Dr. Lines, in his presidential address, and Dr. Ramsay and Dr. Fay, at the plenary session, voiced the general opinion of the Tasmanian Branch, that the community hospital system is the only satisfactory solution of the problem. As Dr. Fay pointed out in his excellent paper, the system is not new and is being widely adopted abroad, especially in the United States of America. Personally speaking, one feels that the intermediate hospital system is not going to prove satisfactory. It is difficult to see how intermediate hospitals will be able to supply the increasingly expensive equipment necessary for investigation and treatment; or, if they do so, to believe that they will be able to carry on at a profit, with such huge overhead costs. If past history is any guide, the running costs of hospitals will increase rather than decrease. It is surely a better policy to allow all to have access to the best and most up-to-date methods of investigation and to have these centralized to reduce costs and to supply accommodation in keeping with the patients' means. The class with moderate means would thus not be penalized, as they are at present in some of the States, from access to the best equipped hospitals; and these and the wealthier classes would, by paying higher fees, largely contribute to the upkeep of the public hospitals. At the same time, any registered medical practitioner should have the right to attend and to charge his own patients in such wards, subject to the approval of the hospital board,

and the profession should have representatives on every hospital board. Increasing specialization seems to be inevitable, and eventually it is probable that each patient will be under the care of one or more specialists for the full investigation of his condition.

It was a great pity that time was not available for a full discussion of the propositions read by the representatives of the State Branches. This was especially unfortunate in view of the presence of the recently formed Federal Council, which replaced the Federal Committee last year. This is now a corporate body with far more extended powers than its predecessor, which was an advisory body only. The Federal Council can initiate and carry into effect policies beneficial alike to the profession and the public. It has the power of active leadership and will act as a link between our Branches and the Parent Association in Great Britain. This Council must take a leading part in the evolution of a general hospital policy for the British Medical Association in Australia. We in Tasmania might have been saved many barren years, detrimental alike to the profession and the public, had the Federal Council existed when the split occurred on the question of hospital policy between the Tasmanian Government and this Branch of the British Medical Association. It is to be hoped that the selected representatives of the Branches who met during Congress to discuss the question of a common hospital policy, were able to present to the Federal Council for consideration certain broad principles that may be acceptable to all Branches. I would like to congratulate Dr. Shugg and Dr. Gibson on their appointment to the Federal Council for the ensuing year. This body is destined to take an increasingly important part in the public life of the Commonwealth, and it thus behoves all Branches to nominate to the Council only our ablest and most experienced men.

Obstetrics and Gynæcology.

Being especially interested in obstetrics and gynæcology, I hope I shall be forgiven if I dwell more particularly on the work of this section. Dr. Schlink's paper, "Prophylaxis of Uterine Cancer", was one of first importance. There is no doubt that some practitioners are not alive to the importance of thorough investigation of all cases, and especially of those occurring in patients within the cancer age, who complain of irregular bleeding or suspicious discharge, especially a watery discharge from the vagina. It is not only the public who have to be educated on this matter.

Dr. Schlink emphasized the large proportion of patients belonging to number 2 or more advanced groups in Döderlein's classification who are admitted to the Royal Prince Alfred Hospital gynæcological wards. Our experience at the Launceston Public Hospital is unfortunately similar to Dr. Schlink's, and it is very rarely that a Group 1 case is sent in; a few Group 2 cases are seen, but the great majority belong to Groups 3 and 4, and the outlook in such is practically hopeless. Of course, many of these women have not consulted a doctor until driven to do so by the urgency of their symptoms, but here and there we find women who have been to a doctor for advice, and the possible serious nature of the case has not been realized until the chances of a cure had become more remote. While in Sydney last year I was very impressed with the teaching methods in the gynæcological clinics at the Royal Prince Alfred Hospital. It is a great advance on the teaching in my student days, and if all schools are as thorough we shall see in future a gradual rise in patients belonging to Groups 1 and 2 admitted to hospital for treatment and a corresponding fall in those of Groups 3 and 4.

It seems to be generally held that a biopsy of cervical tissue, especially if a diathermy wire loop is used, is not contraindicated, and the same risk is not incurred as in the removal of a lump from the breast.

At the Launceston Public Hospital we have lately been using Schiller's test of staining cervices with Lugol's solution as a routine measure, and hope to find it of value in discovering some cases of cancer in the very earliest stage—the so-called pre-cancerous condition. The colposcope introduced by Hinselmann, of Hamburg, is being used at the Royal Prince Alfred Hospital, Sydney, and promises to be of use in diagnosing early cases of cancer of the cervix. It gives a high magnification and early atypical changes at the junction of vaginal and cervical mucosa, possibly the precursor of cancer, may be detected. An even better instrument is being tried out in the United States of America.

One has read various articles in which it is advised that as prophylaxis against cancer all torn cervices should as a routine measure be repaired immediately after the completion of the third stage of labour. While theoretically this may be a sound proceeding and one that might be safely carried out in first class hospitals, I think few obstetricians care to risk infecting the upper vaginal passage and uterus under conditions usually found in maternity homes and private houses.

Unfortunately the use of radium before cancer has developed does not seem to give protection against the disease developing at a later date. It is open to discussion, therefore, whether it might not be advisable to amputate the cervix of every parous woman who has lacerations and erosion of the cervix, once she has reached the menopause. It is an operation attended with little risk or pain, and would save many lives and untold suffering and misery.

As regards treatment, it was generally accepted by the Section that in cancer of the body of the uterus intrauterine application of radium, followed in three weeks by panhysterectomy, is the treatment of choice. In cancer of the cervix, however, there still seems to be a wide difference of opinion between surgeons and radiologists. In a

few years the New South Wales Cancer Research Committee will have some very valuable information on the question. The Royal Prince Alfred Hospital is combining radium and operation, a Wertheim operation being done three weeks after radium treatment. A series of microscopic sections are cut across the whole uterus in one piece, and the effects of radium on the cancer tissue can be demonstrated. These sections are shown by the pathologist to the students at the clinic a week after operation. The Sydney Hospital staff is using radium alone, while at Saint Vincent's Hospital radium and X radiation are combined. In all three hospitals a keen follow-up system is carried out. At a future date it will be interesting to see whether too much is claimed by radiologists or whether surgery will have to surrender another stronghold to radiation. The Royal Prince Alfred school is of opinion that even if results are equally good over, say, a five years period, there is still danger of cancer returning, even at a remote period, in nests of cancer cells, "embalmed areas", which have become latent and apparently quiescent, but not totally destroyed by radiation.

The really important lesson for us all to learn from this paper, and from the discussion following, and from many recent publications, is that we are not likely to advance much further with treatment in the near future, and that we should concentrate our efforts in preventive measures. All chronic cervical lesions at or after the menopause should be dealt with by surgery, preferably low amputation of the cervix, or in certain cases by the electro-cautery. It is also questionable whether a subtotal hysterectomy should ever be done in a parous woman without a thorough coning out of the cervix either from above or below.

Every doctor should, without unnecessarily alarming them, explain to his women patients who have reached the menopause those signs and symptoms which should cause them to seek advice without delay. During his routine examination, which should include the breasts, he should make it his business to examine the cervix visually, and if chronic lesions are found, treatment without undue delay should be urged.

Dr. Worcester's paper on the treatment of abortion was, I think, opportune, and I was particularly interested in the discussion that followed it. Unfortunately there was no one present who spoke in favour of the Melbourne teaching of non-interference in septic cases, except by intrauterine glycerine injections. The meeting was unanimous in advising careful and early curettage of the uterus in all cases with retained secundines, with a few exceptions, such as in gravely ill patients, or those in whom such complications as acute pelvic cellulitis, acute salpingitis, and so on, had arisen. One would like to have heard someone speak for the other method of treatment. Two years ago I adopted the Melbourne teaching and treated all patients conservatively, using intrauterine application of glycerine only, and I have definitely come to the conclusion that much more harm is done by leaving dead necrotic tissue in the uterus than may occasionally be done during a careful removal by a blunt curette. Glycerine is a useful adjunct and can be used after curettage, but, though it has a strong hygroscopic effect and encourages a fluid discharge, I do not find that it has much effect in removing the tissue itself, which remains in the uterus as a source of toxæmia and increases the morbidity, if not the mortality, in these cases.

Radiology.

I should like to congratulate the Section of X Ray and Electrical Therapy on the provisional formation of an Australian Association of Radiology. It is proposed to form a provisional Federal Council to draw up articles of association at Canberra during the April Cancer Conference. In the study of our clinical problems X rays are becoming increasingly important, and new methods of investigation in this field are being constantly brought forward. The day has long passed when a practitioner could do adequate work with a small plant at his surgery. Still more important is it that radium and, of course, X ray therapy, should be entirely in the hands of men specially trained for such work. The rival claims of the radiologists and the surgeons in certain fields have yet to be settled. Time only will show the limitations and advantages of one as opposed to the other, but it is certain that in the next few years we shall be able to say with confidence which treatment is most suitable for the case under consideration. The formation of this Association of Radiology will lead to the best technique being evolved and standardized in all radiation treatments. There is still much to be learned in the treatment of cancer by radium. Much of the work has been carried out by surgeons with no special training in, or enthusiasm for, radium. Dosage, filtration, methods of application *et cetera* in gynæcological work have been much improved in recent years and better results have been obtained. The enthusiasm of earlier radiologists who claimed too much, received a check, and there is still much debatable ground over which radiologists and surgeons may argue, but slowly and surely the field of work for each is being defined. There is no doubt that the best results are obtained when radiologists and surgeons work together. At the Launceston General Hospital the gynæcologist and radiologist consult together over every case in which radium treatment might be indicated, and both are present whenever radium is used, the radiologist applying the radium after the surgeon has done his part. In this way, both become familiar with the technique, and together study the progress of such cases at the cancer clinics.

Cancer.

The discussion on cancer suffered in the same way as that on the hospital problems. All the morning was taken up by the papers, and little time

was left for general discussion. In particular, Dr. Clendinnen's paper on radium deserved a great deal of attention and no doubt would have received much criticism from the surgeons. Dr. Clendinnen's experience with radium and X ray therapy is second to none in Australia; he is one of the pioneers of this branch of therapeutics in the Southern Hemisphere, and as such must be listened to with respect. His results from radiation treatment of secondary glands in the neck are rather outstanding, as were those obtained by the use of buried, heavily filtered radium needles in bone or cartilage. He made another and perhaps less unexpected plea for the use of radiation treatment as opposed to surgery in breast cancer. Admittedly the results of surgery for this condition are disappointing. If radiation could equal or even approach surgical results, and if the patient could be saved the mutilating operation involved in a radical removal of the breast, a great advance would be achieved.

Mr. Zwar and other speakers urged that women should be encouraged to have regular systematic examinations of the breasts when they reached the cancer age, since it was only in early cases that good results, either by surgery or radiation, or both combined, could be obtained. The need for such regular examinations of the uterine organs was emphasized by speakers in the gynæcological section, and I would again urge the great importance of visual examination of the cervix and of biopsy in all suspicious cases. This leads one to refer to the public education on cancer. We have been subjected to some degree of criticism for undertaking such a campaign, most of it puerile, but many lay people consider it wrong to increase cancerphobia and to upset the peace of mind of the timid. Undoubtedly it is true that many people are frightened by the name cancer and shrink from presenting themselves for examination until compelled by urgent symptoms to do so. But this makes it all the more important that we should do all in our power to lower the mortality, and so in time lessen the dread of this scourge. We can do this only by educating the public so that all should be alive to the danger signals. With prenarcosis we have greatly diminished the fear and discomfort of anæsthetics and could we promise a reasonable chance of cure by radiation alone there would be little difficulty in persuading all people who have reached the cancer age to have periodic examinations carried out. Prevention should be the ideal at which we aim, but in the present state of our knowledge is difficult of attainment. We should at any rate by this education be given a chance to discover it in its early and curable stages.

We began our educational campaign in the north of the island last year when the Northern Tasmanian Anti-Cancer Committee was formed with the following objects in view:

1. The provision of adequate methods of treatment.

2. An organization, preferably permanent, for the systematic education of the public in regard to the cancer menace.

3. A consultative clinic for the examination and treatment of patients unable to pay.

4. Provision for adequate social services and for an efficient follow-up system.

Articles on cancer in all its aspects suitable for public consumption were prepared by medical men and were submitted for approval to the Anti-Cancer Committee Council and published in the Press. Short weekly lectures prepared by medical men were given over the wireless by the Commonwealth Public Health Officer. After a few weeks the educational aspect was shelved for the time being and the efforts of the committee were concentrated on collecting funds for the purchase and installation of the deep therapy equipment. Tenders have been called for and are now in the hands of the Northern Tasmanian Anti-Cancer Committee. It is now timely to recommence work for the second objective, that is, public education, but before we do so we feel that the new campaign should be organized to embrace Tasmania as a whole. This matter was discussed at a meeting of the Branch last year, but it was decided to postpone such activities until after Congress, and no doubt the new Branch Council will do all in its power to forward this work. As an initial step, the formation of a central anti-cancer campaign committee for the whole island is required, and it is hoped that the new Branch Council will assist in the formation of this body. It will thus be making an early response to the motion put to the plenary session on cancer by Dr. E. S. Meyers and unanimously carried:

> That in the opinion of this Congress the time has arrived for a more active cooperation between governments, medical organizations, scientific bodies, and the general public, in the prosecution of the Australian campaign against cancer.

THE TOXICOLOGY OF DUBOISIA AND ANTHOCERCIS.

By JOHN MACPHERSON, M.A., B.Sc., M.B., Ch.M. (Sydney), *Lecturer on Materia Medica, University of Sydney.*

THE chemistry of the species of *Duboisia*, belonging to the great family Solanaceæ, was described in detail in THE MEDICAL JOURNAL OF AUSTRALIA of July 28, 1917. The Australian Encyclopædia (1927) states that all three species yield atropine, hyoscyamine and other mydriatic alkaloids. This is not quite accurate. The corkwood or native elm (*Duboisia myoporoides* R. Brown) is an evergreen shrub or small tree, reaching a height of fifteen to twenty feet and occurring in forest lands from near the Shoalhaven River along the eastern area of New South Wales to near Cape York in Queensland. Its habitat extends also to New Guinea and New Caledonia. It has small bell-shaped flowers, white in colour, with

purplish stripes, and a succulent globular fruit or "berry". The leaves reach the length of ten centimetres (four inches). The active principle has been termed "duboisine" and is mostly obtained from the leaves and twigs, but is found also in the bark. The leaves contain rather less alkaloid during the flowering season than later. November and December are said to be the best months for collecting the leaves for their alkaloidal content. The leaves have a somewhat sickly, narcotic odour, with a bitter taste, but not so burning or acrid as those of *Duboisia hopwoodii*. The active principle ("piturine") of the latter has a far more powerfully irritant effect on the eyes and respiratory passages than has duboisine. The earlier investigations of duboisine gave conflicting results, owing to the fact that sometimes the bark was used for examination, and the bark contained in addition to duboisine a resin-like body and other constituents. Duboisine, as a sulphate, is largely used in the United States of America and the Continent of Europe for therapeutic purposes, whereas it is almost unknown in Australia, which is its country of origin. A profitable industry in Australia is thus being almost neglected.

Duboisine is not a simple chemical compound, but is a mixture of alkaloids, and is generally stated to be a varying mixture of hyoscine and hyoscyamine, chiefly the latter. But it may be almost pure hyoscine. The proportions vary according to the soil and the conditions of the plant from which it is extracted. The precise constituents of duboisine are: l-hyoscyamine, nor-hyoscyamine (pseudo-hyoscyamine) and l-scopolamine (hyoscine). The dose of duboisine recommended in therapeutics is 0·5 to 1·0 milligramme (one one-hundred-and-twentieth to one-sixtieth of a grain), but even 2·0 to 3·0 milligrammes (one-thirtieth to one-twentieth of a grain) have been used by hypodermic injection. Weber recommended, in delirious mania, the administration of even two mils (30 minims) of a 0·2% solution—equal to nearly 4·0 milligrammes (three-fiftieths of a grain).

Toxicology.

The Reverend Dr. Wools, in 1861, pointed out that this plant possessed powerfully intoxicating properties. He had known an instance in which giddiness and nausea had arisen from remaining in a close room in which branches of it were placed. He said that the odour was faint, but sickly and not nearly so intense as that of *Duboisia hopwoodii*. In both species the active principle is to a certain extent volatile. The aborigines were wont to make holes in the trunk of the tree and fill such holes with water, which, when drunk on the following morning, produced stupor. Branches were thrown into pools to intoxicate eels and bring them to the surface to be readily captured. It has been stated that white people, trying the method of the natives, quickly succumb and remain in a stupor for hours. The pharmacological actions and therapeutic uses of duboisine are those of belladonna, strammonium, hyoscyamus and *Datura fastuosa* or their active principles. Duboisine generally is less irritant to the conjunctiva than atropine, but it may cause irritation and hyperæmia, with a stinging sensation. Erythema of the lower eyelids is less likely to be evoked than with atropine. However, follicular conjunctivitis to a marked degree and an inflammatory condition of the lower lids and adjoining area of the cheek, closely simulating erysipelas, have followed the use of eye drops. The use of eye drops requires caution, as toxic constitutional symptoms have followed such drops of a strength of less than 1%. When given orally or by hypodermic injection, duboisine is more liable than atropine to cause toxic symptoms, and such cases as display idiosyncrasy to atropine are apt to evince the same intolerance to duboisine. The symptoms of poisoning are: dryness of the mouth, throat and other mucous membranes, with intense thirst; there may be a soapy taste in the mouth or loss of taste. Rarely, for some unexplained reason, there is profuse salivation. When it is given before meals, there may be disorder of ensuing digestion. When it is given to hysterical or insane patients there has been noticed vomiting of part of the food without nausea. Duboisine paralyses vagus nerve endings like atropine. It checks (rarely increases) perspiration. It impairs eyesight by paralysing accommodation. It sometimes diminishes urinary secretion. Duboisine is not generally a cerebral excitant, like atropine, but rather a calmative, sedative and hypnotic. Occasionally, however, delirium has been noted. Symptoms referable to the central nervous system are headache, giddiness and drowsiness or loss of muscular control. In frogs, tetanus has been observed.

In June, 1933, a male child, aged two years, died at Bellingen from eating the leaves on the afternoon prior to his death. In January, 1934, an aboriginal child was also poisoned, but not fatally, near Bellingen, from eating the bark of the tree. Some years previously two children on the Richmond River were seriously poisoned, but recovered. Their toxic manifestations were delirium with nervous and muscular derangement. This tree was recently reputed to have caused the deaths of three heifers and a steer. It is very dangerous to stock which eat the bark. It has been suggested that some deaths in stock, attributed to snake bite, may have been due to eating this plant.

In treating cases of poisoning the stomach must be emptied by such emetics as sulphate of zinc or of copper or mustard. Morphine may be used to combat nervous disturbances as they arise. It is said that the action of duboisine on the heart is antagonized by muscarine; but this is not practicable as an antidote.

Pituri.

Duboisia hopwoodii F. von Mueller yields pituri, called also pitchery, petcheri or bedgery, with other variants in spelling. This plant occurs

in the central areas of Australia, from the Darling River and Barcoo in north-western New South Wales and south-western Queensland across to Western Australia. It was described by von Mueller in 1874 from specimens brought back from the unhappy Burke and Wills expedition in 1861. An alkaloidal constituent was termed by Gerrard "piturine". This has now been definitely identified as nicotine, although A. Liversidge considered it to differ both from nicotine and pyridine. Pituri was a drug of addiction among the native blacks. The parts used were the twigs and fragments of leaves, mixed with twigs and leaves of a species of acacia or ashes of eucalyptus leaves or threads of native flax. Pituri is brownish in colour, with a tobacco-like odour and a burning, acrid taste. The dust causes sneezing. Pituri has a definite mydriatic action. The natives chewed the rolls or bolus, but did not smoke it. When not in use it was placed over the top of the ear. The aborigines craved for pituri, and in areas where it did not grow bartered other commodities for it. On ceremonial occasions the bolus was passed from one person to another, each masticating it before its return to the owner. The manifestations of pituri chewing do not seem to accord entirely with those resulting from the use of tobacco in the white races. Pituri invigorates the natives in their long marches in the desert and excites their courage in warfare. Some have said that a large dose infuriates them, but others have denied this. White men who have smoked pituri say that it has a soporific effect, quickly inducing sleep, or may cause headache and rapid intoxication. Amongst the aborigines as a rule only older men used it, not the younger ones or women. Sometimes, however, women were permitted its use.

Sir Edward Stirling, on the Horn exploring expedition to Central Australia, did not see pituri used as a narcotic, but only to contaminate water holes and so enable the capture of fish, birds and beasts. Pituri is an emu and camel poison. Leaves were placed by the blacks in water to stupefy emus, which, after drinking, were easily caught and killed. Sir Baldwin Spencer wrote that in Central Australia the leaves were pounded in water and the infusion, placed in a wooden vessel, was put where the emus would drink it in the scrub; or the leaves were placed in pools of water frequented by emus. After drinking, the emus became stupefied like men intoxicated by alcohol and fell an easy prey to aboriginal spears.

Duboisia leichhardtii F. von Mueller was discovered by Leichhardt. It occurs in the eastern States of Australia, particularly Queensland, and occupies the area between the coast and the central regions of Australia. The leaves contain l-hyoscyamine, nor-hyoscyamine and l-scopolamine. J. M. Petrie found fresh leaves to contain 0·28% of mixed alkaloids, from 0·5 gramme of which he separated 0·1 gramme of l-hyoscyamine, 0·2 gramme of nor-hyoscyamine and 0·1 gramme of scopolamine.

Anthocercis.

The genus *Anthocercis* is closely allied to *Duboisia* and has seventeen Australian species. The habitat extends over the greater part of Australia, including Tasmania. Species occur in every State, particularly in Western Australia. *Anthocercis viscosa* R. Brown, of Western Australia, has a yellow, liquid, oily alkaloid, heavier than water, and known as "anthocercine". It is alkaline in reaction and has an agreeable odour. Probably the active principle of the genus is hyoscine or hyoscyamine, or both. The plants have a powerfully mydriatic effect. *Anthocercis littorea* Endlicher is another Western Australian species with yellow flowers. Children in Western Australia have been poisoned by this plant, the symptoms resembling belladonna poisoning. Dr. Moffit, of Greenough, has reported the case of a male child, aged five years, who chewed the plant. After coming home about tea time he suddenly fell down. There was incoherent speech and wild muttering delirium. The arms were thrown aimlessly about. The mouth and throat were dry, the pupils were dilated, the pulse was strong, and there was no rise of temperature. After treatment with potassium bromide and chloral hydrate the child slept for six hours and recovery ensued. Further references to *Anthocercis* are given by Professor J. Burton Cleland in the *Australasian Medical Gazette* of June 20, 1914.

Nicotiana.

Nicotiana suaveolens Lehmann is the native Australian tobacco. It is a very variable species, ranging across the continent, but chiefly occurring in the interior. Several sub-species or varieties are described, which some authorities would raise to definite specific rank. The flowers are white or greenish and sweet-scented. I can find no record of the aborigines employing this plant, but colonists in the early days used the leaves as tobacco. Stock eat it readily and with impunity, if mixed with other herbage, but, if eaten alone in considerable quantity, it might prove toxic on account of its nicotine content.

OBSERVATIONS ON PHOSPHATE EXCRETION IN NORMAL AND TOXÆMIC PREGNANCY.

By VERA I. KRIEGER, M.Sc.

(From the Biochemistry Department, University of Melbourne, and the Women's Hospital, Melbourne.)

IN 1929 Brain and Kay[1] published a method for the estimation of renal function based on the measurement of the excess phosphate excreted in the urine during the hour following intravenous injection of a fixed amount of sodium glycerophosphate. This method was based on the suggestion of Eichholtz, Robison and Brull,[2] that the inorganic phosphate of urine is derived from the hydrolysis of phosphoric esters in the blood plasma

by the enzyme phosphatase in the kidney tissue. Although later work seemed to indicate that this theory was incorrect, the practical results of the test seem to be very satisfactory.

Brain and Kay[1] showed that in the terminal stages of chronic nephritis little, if any, extra phosphate was excreted. They also claimed the test to be as useful as either the urea concentration or phenolsulphonephthalein test, and stated that in certain cases, where divergence occurred, the result of the glycerophosphate test indicated the condition of the kidneys more reliably than either of the other tests.

Since the condition of the kidney is one of the factors of marked importance in toxæmias of pregnancy, it was thought advisable that the glycerophosphate test should be investigated in a series of normal women, normal pregnant women and toxæmic pregnant cases (Tables I and II). The test was compared with the blood urea and urea concentration tests. An analysis of the results

TABLE I.

Comparison of Phosphate Test with Blood Urea and Urea Concentration Tests in a series of Normal and Toxæmic Pregnant Women.

Most of these patients were investigated during the month prior to delivery.

Case Number.	Phosphate Test. Milligrammes of Phosphorus Excreted per Hour.	Blood Urea. Milligrammes of Urea per 100 Cubic Centimetres of Blood.	McLean's Urea Concentration. Percentage.	Type of Patient.
7	0	24	1·9	Chronic nephritis.
22	0	102	1·4	Chronic nephritis.
27	0	58	0·6	Chronic nephritis.
30	0	39	1·8	Nephritic toxæmia.
71	0	23	1·3	Chronic nephritis.
84	0	22	3·0	Normal.
122	0	17	2·6	Normal.
108	10	36	1·7	Chronic nephritis.
109	14	22	1·5	Chronic nephritis.
90	15	18	1·9	Chronic nephritis.
65	17	16	1·8	Chronic nephritis.
102	17	30	2·0	Albuminuria (severe).
20	21	25	—	Eclampsia (9 fits).
3	28	44	2·7	Normal.
118	29	4	—	Chronic nephritis.
123	32	36	3·4	Normal.
111	34	16	1·9	Chronic nephritis.
32	34	25	1·7	Chronic nephritis.
14	36	20	1·7	Chronic nephritis.
60	36	43	3·1	Nephritic toxæmia.
105	36	36	1·6	Albuminuria.
19	37	42	2·7	Nephritic toxæmia.
17	40	23	2·6	Albuminuria.
127	40	17	—	Normal.
21	41	44	3·2	Albuminuria.
70	41	21	2·0	Nephritic toxæmia.
29	44	43	2·0	Nephritic toxæmia.
36	45	24	1·6	Chronic nephritis.
130	46	—	—	Normal.
113	49	16	2·5	*Ante-partum* hæmorrhage.
116	50	18	1·4	Normal.
57	53	38	2·5	Pre-eclampsia.
120	56	—	—	Normal.
40	58	23	2·7	Albuminuria.
63	58	28	2·8	Chronic nephritis.
31	61	24	3·1	Albuminuria.
15	64	60	—	Albuminuria.
117	64	35	0·9	Normal.
125	64	17	2·2	Normal.
2	70	42	3·9	Albuminuria (severe).
56	72	25	—	Normal.
124	76	18	3·3	Normal.
4	76	43	2·4	Albuminuria.
6	78	47	—	Eclampsia.
119	78	—	—	Normal.
140	80	19	2·1	Normal.
1	83	—	—	Normal.
87	86	43	1·1	Chronic nephritis.
132	86	—	—	Normal.
126	93	14	—	Normal.
121	96	—	—	Normal.
5	102	58	2·9	Eclampsia (2 fits marked albuminuria).
12	105	36	2·9	Chronic nephritis.
26	106	24	2·6	Albuminuria.
104	109	34	3·5	Normal.
137	111	23	—	Albuminuria.
11	112	40	2·0	Normal.
80	115	24	2·0	Chronic nephritis.
131	116	—	—	Normal.
52	118	40	3·5	Normal.
82	121	24	2·6	Albuminuria.
17	126	26	2·8	Albuminuria.
91	135	14	3·3	Pyelitis and chronic nephritis.
133	143	13	3·1	Normal.
134	160	14	3·2	Normal.
128	252 (At 7 months. Value fell to 16 milligrammes of phosphorus per hour four days after delivery.)	—	—	Normal.

TABLE II.

Comparison of Phosphate Test with Blood Urea and Urea Concentration Tests in a series of Normal and Toxæmic Women during the two weeks following delivery.

Case Number.	Phosphate Test. Milligrammes of Phosphorus Excreted per Hour.	Blood Urea. Milligrammes of Urea per 100 Cubic Centimetres of Blood.	McLean's Urea Concentration. Percentage.	Type of Patient.
86	0	24	1·4	Chronic nephritis.
101	21	62	2·1	*Intra-partum* eclampsia.
107	25	—	2·0	*Intra-partum* eclampsia.
33	30	87	1·0	Chronic nephritis.
54	32	33	3·8	Nephritic toxæmia.
94	36	17	2·5	Albuminuria.
10	54	49	4·3	Normal.
88	55	27	3·5	Normal.
28	56	30	1·9	Eclampsia (9 fits).
39	61	27	1·7	Chronic nephritis.
		62	39	2·8	Normal.
76	70	31	3·0	Albuminuria.
114	74	27	1·4	Chronic nephritis.
38	76	38	3·1	Albuminuria.
51	77	—	—	Normal.
68	79	33	1·3	Eclampsia (4 fits).
41	81	18	2·9	Albuminuria.
85	88	25	2·6	Albuminuria.
67	89	25	2·0	Chronic nephritis.
89	93	35	1·6	Albuminuria.
83	93	23	2·8	Albuminuria.
59	94	—	2·1	Pre-eclampsia.
100	96	27	2·4	Albuminuria.
25	98	21	1·9	*Ante-partum* eclampsia.
37	98	31	3·3	Nephritic toxæmia.
64	98	36	2·7	*Post-partum* eclampsia (7 fits).
113	102	23	2·2	Nephritic toxæmia.
38	103	31	2·9	Albuminuria.
55	104	33	3·5	Albuminuria.
23	106	26	—	Chronic nephritis.
24	108	47	1·8	*Intra-partum* eclampsia (2 fits).
61	113	39	3·6	*Post-partum* eclampsia (2 fits).
62	115	9	3·3	Normal.
69	116	20	2·1	Nephritis.
13	117	22	1·6	Albuminuria.
110	118	14	1·9	Nephritic toxæmia.
115	119	17	1·7	Chronic nephritis and *ante-partum* eclampsia.
9	124	—	4·0	Albuminuria.
107	128	17	1·4	*Intra-partum* eclampsia.
112	131	—	—	Pre-eclampsia.
18	132	42	1·9	Chronic nephritis.
138	132	22	3·2	Albuminuria.
95	132	21	4·1	Albuminuria.
103	133	26	2·1	Albuminuria (severe).
72	135	39	2·3	Nephritic toxæmia.
8	137	—	4·8	Albuminuria.
15	137	37	3·2	Albuminuria.
106	140	24	2·4	Albuminuria.
77	147	—	—	*Ante-partum* eclampsia.
93	150	17	2·1	Secondary anæmia of pregnancy.
98	150	23	3·0	Chronic nephritis.
7	157	—	—	Chronic nephritis.
73	162	22	2·1	Pre-eclampsia.
78	187	19	2·7	Pre-eclampsia.
74	192	33	1·5	Chronic nephritis.
99	195	18	2·1	Pyelitis.
135	202	—	3·0	Albuminuria.

TABLE III.

Type.	Non-Pregnant Group.				Pregnant Group.							
					Before Delivery.				After Delivery.			
	Number of Cases.	Percentage Cases with Excess Phosphate Excretion.			Number of Cases.	Percentage Cases with Excess Phosphate Excretion.			Number of Cases.	Percentage Cases with Excess Phosphate Excretion.		
		Less than 90 milligrammes per hour.	90 to 150 milligrammes per hour.	More than 150 milligrammes		Less than 90 milligrammes per hour.	90 to 150 milligrammes per hour.	More than 150 milligrammes		Less than 90 milligrammes per hour.	90 to 150 milligrammes per hour.	More than 150 milligrammes
Normal ..	6	0	100	83	30	70	30	13	9	44	56	33
Albuminuria	—	—	—	—	9	77	22	0	19	39	62	0
Eclampsia ..	—	—	—	—	2	1 case	0	1 case	14	48	52	0
Nephritic toxæmia ..	—	—	—	—	6	83	17	0	6	17	85	0
Chronic nephritis ..	—	—	—	—	15	80	20	0	12	42	56	17
All Cases ..	6	0	100	83	62	74	24	2·7	60	40	60	11
Total toxæmic cases ..	—	—	—	—	32	78	22	0	51	39	61	8

showed no correlation between these renal function tests and the phosphate excretion. In several cases high blood urea, low urea concentration and low phosphate excretion seemed to confirm the agreement of the test with those already in common use. On the other hand, many cases showing normal blood urea, normal urea concentration and normal clinical history gave very low values for the excess phosphate excretion. Again, many cases with low figures for the urea concentration test and with a normal blood urea showed much higher excess phosphate excretion than other cases where the renal function, as assessed by the urea concentration test, was very much more satisfactory.

These results therefore show that this test seems to have no value whatever as a test for kidney efficiency in pregnancy. Even in normal pregnancy about 18% only of the patients examined showed an excretion of excess phosphate as high as 150 milligrammes per hour, which is the value quoted as the minimum excess excretion for normal kidneys (Table III).

In a small group of normal non-pregnant women the average excess phosphate excretion was 169 milligrammes per hour. only one figure falling below the minimum figure of 150 milligrammes per hour. In the toxæmic group only 5% of the cases gave values above the minimum.

There was only a 62% agreement between the urea concentration test and the excess phosphate excretion when 90 milligrammes per hour excess phosphate excretion was arbitrarily used as the minimum value.

A close examination of the results obtained during the investigation reveals the interesting fact that there is a marked decrease in the excess phosphate excretion prior to delivery. The majority of the tests performed before delivery were made during the last month of pregnancy. In the normal pregnant group 70% of women gave values of less than 90 milligrammes per hour excess phosphate excretion as compared with 78% of the total number of toxæmic patients examined. In the nephritic toxæmias about 80% of subjects showed excess excretion below 90 milligrammes per hour (Table III). In the pre-delivery groups 13% of the normal subjects exceeded 150 milligrammes per hour, but none of the toxæmic cases approached this figure.

In Table IV the results have been arranged in three groups: (i) values below an arbitrarily chosen 90 milligrammes per hour, (ii) between 90 and 150 milligrammes per hour, and (iii) those above 150 milligrammes per hour. This grouping shows the marked retention of phosphate in the pre-labour period, and a definite increase after delivery in about 50% of the cases, whether the subjects were normal or toxæmic. Even in normal pregnancy only about one-third of the subjects reach the 150 milligrammes per hour excretion rate after delivery. In toxæmic pregnancy there are less than 10% of the subjects with the normal excess excretion rate after delivery.

TABLE IV.

Excess Phosphate Excretion.	Normal.		Toxæmic.	
	Pre-delivery.	Post-delivery.	Pre-delivery	Post-delivery.
	%	%	%	%
150 milligrammes per hour..	13	33	—	8
90 to 150 milligrammes per hour	17	27	22	53
90 milligrammes per hour..	70	40	78	39

In certain cases tests were performed on the same patient before and after delivery. In this group (Table V) 90% of subjects show marked increase in the excess phosphate excretion after delivery. Only 23% of these showed values above 150 milligrammes per hour after delivery.

These observations raise the question as to why, even in normal pregnancy, there is this great decrease in excess phosphate excretion during the later weeks of pregnancy and this definite rise, although the rise is still far below the level for normal non-pregnant individuals.

The fall before delivery may be due to the demands of the growing fœtus for increased

Table V.

Type.	Case Number.	Before Delivery. Milligrammes per Hour.	After Delivery. Milligrammes per Hour.	
Normal Pregnancy	1	69	178	
	2	86	202	
	3	0·7	132	
	4	252 (28 weeks)	16	4 days after delivery.
Toxæmias (a) Albuminuria	1	17	133	
	2	36	140	
	3	No excess	—	
		109		
	4	126	—	2 months before delivery.
		40	—	7 days before delivery.
(b) Eclampsia	1	21	56	*Ante-partum.*
	2	—	128	*Intra-partum*
			25	
(c) Nephritic Toxæmia	1	41	135	
	2	44	49	
	3	37	102	
(d) Chronic Nephritis	1	No excess	188	
	2	No excess	30	
	3	10	74	

amounts of phosphate for bone formation. After delivery, therefore, the excess phosphate excretion increases, but there is still the need for increased amounts of phosphate in the mother during the lactation period for the production of milk. This may account for the large number of instances in which increased values occur after delivery, although these may be still far below the normal non-pregnant rate of excess excretion.

Conclusions.

The glycerophosphate test for determining renal efficiency, formulated by Brain and Kay, is useless in pregnancy. There is a marked decrease in the excess phosphate excretion after injection of sodium glycerophosphate in both normal and toxæmic pregnancy. There is a definite increase in the excess phosphate excretion following injection of sodium glycerophosphate after delivery in both normal and toxæmic pregnancy. The excess excretion is still below the normal non-pregnant rate. There is a higher rate of excretion among the normal pregnant cases.

Acknowledgements.

I wish to thank Dr. H. H. Martin and Dr. Ormond Green for their help in giving the sodium glycerophosphate injections and collecting the histories of the cases investigated.

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PATHOLOGICAL LESIONS MET WITH IN THE ŒSOPHAGUS DURING POST MORTEM EXAMINATIONS.

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At the Adelaide Hospital 2,700 *post mortem* examinations, mostly carried out by myself, have now been tabulated and, with 300 at the Mental Hospital at Parkside, make a total of 3,000. By dividing the figures by 30 the percentage of occurrence of the various lesions is readily obtained.

Carcinoma of the Œsophagus.

There were 17 examples of carcinomata of the œsophagus out of a total of 361 carcinomata, or including sarcomata *et cetera*, 389 malignant growths of all kinds. Fourteen were in males and only three in females. The youngest patients were 42, 48 and 56 years of age, and nine of the 17 were in their sixties, three patients were in the seventies, one was aged 80, and the age of one was unknown. Common causes of death were septic pneumonia or the formation of abscesses in the lungs, one patient dying from abscesses in the brain which were secondary to abscesses in the lungs.

Submucous Lipoma.

A chronic dement of seventy-nine, who had a malignant condition, perhaps a reticulo-endothelioma, in the glands of her neck, was found to have a submucous lipoma projecting downwards from the anterior wall about 4·3 centimetres (one and three-quarter inches) above the level of the bifurcation of the trachea. The attachment was sloping, the shape cylindrical, with the lower half inch projecting downwards with a conical end, and the diameter was about 1·2 centimetres (half an inch). As far as is known, this considerable polypoid structure gave rise to no inconvenience during life.

Other Simple Growths.

A woman of forty-two had a mass of soft tissue five centimetres (two inches) in diameter, containing a multilocular cystic cavity, behind the lower part of her œsophagus, together with irregular syphilitic cirrhosis of the liver and a very large spleen. A woman of sixty-four, suffering from subacute melancholia, had two or three adenomatous polyps, twelve by six millimetres in size, in the lower part of the œsophagus.

Vascular Lesions.

Varicosities were sufficiently prominent to be noted in two cases, that of a male of sixty-one who died from hæmorrhage with much blood in the stomach and intestines, and of a male of seventy-one. Hæmorrhagic streaks occurred in a male, aged fifty-eight, who was convalescing from the typhus-like disease described by Dr. F. Hone. An insane patient, aged eighty, had small petechial hæmorrhages and congestion at the lower end of the

œsophagus, with his stomach and upper part of the duodenum filled with tarry altered blood.

Leucoplakia et cetera.

A woman of seventy-four with a diaphragmatic hernia showed leucoplakic patches, and a man of forty-two, small warty plaques in the œsophagus.

Infective Conditions.

A woman of forty-nine had a small abscess in the œsophagus, early pericarditis with renal fibrosis and cardiac hypertrophy. A male of twenty-two, with large white kidneys from subacute nephritis and some renal œdema and uræmia, showed acute ulceration of the upper part of the œsophagus. A woman of twenty-six, suffering from a profound anæmia of pregnancy with ulceration of the ileum, had a sloughing œsophageal mucosa. A male of seventy-three, with a tumour of the roof of the lateral ventricle in the brain, and bedsores, had an ulcer in the œsophagus near the ary-epiglottidean fold, which was thought to be due to his acute decubitus. A woman of fifty, suffering from chronic interstitial nephritis, died from a *Staphylococcus aureus* infection in which there were much congestion and a membranous exudate in the œsophagus, congested bronchi, hæmorrhagic foci in the lungs, and purulent pericarditis.

Syphilitic Lesions.

A woman of seventy had gummatous syphilitic aortitis and pericarditis which had led to adhesions to the left lung and œsophagus, and ulceration of the œsophagus at the bifurcation of the trachea, with resulting fatal hæmorrhage. An acute melancholic male patient, aged thirty-nine, who gave a positive Wassermann reaction, had discoloured blackish patches at the commencement of the œsophagus, together with diffuse pneumonic consolidation.

Diverticula.

Diverticula were noted in seven cases, and five at least of these appeared to be the result of adhesions of a tuberculous gland in the neighbourhood of the bifurcation of the trachea.

Stricture.

A woman of twenty-eight, who died from exhaustion, had a somewhat doubtful narrowing with congestion, but without thickening at the commencement of the œsophagus.

Achalasia of the Cardiac Orifice.

Three patients had what was probably achalasia of the cardiac orifice, which had given rise to hypertrophy of the muscular wall of the lower part of the œsophagus, the hypertrophy tapering off upwards. In these cases there was no dilatation of the œsophagus immediately above the cardiac orifice, which presumably does not open when the peristaltic wave reaches it. In all probability these were cases of achalasia where the hypertrophied muscular wall of the œsophagus was still capable of eventually passing the contents through the passive orifice, and dilatation, indicating failure to do this, had not yet set in. Since the hypertrophy is greatest at the junction with the stomach, it is hard to see how this portion could dilate or pouch in any of these cases. One was in a man of sixty-four, with a small fleshy fibrotic liver, perhaps secondary to old hydatid disease, ascites and double hydrothorax. The next case is of considerable interest, being in a male mental patient who died from chronic amœbic dysentery with necrosis and perforation of the caecum. Difficulty had been experienced in getting this patient to take his medicine, which included bismuth. He seemed to have difficulty in swallowing it. By putting the medicine into his mouth and tickling him under the chin the patient was able to swallow successfully. At the *post mortem* examination there was much hypertrophy of the lower end of the œsophagus, whose mucosa was jet black in colour, apparently from oxidative changes in the bismuth, which had been frequently retained in that part. The third case, that of a feeble-minded paranoid male patient, aged sixty-four, with a carcinoma in his pancreas, showed hypertrophy of the lower part of the œsophagus. The fourth case, that of a man of sixty-three, with carcinoma of the liver and carcinomatous peritonitis, showed a hypertrophy of the lower part of the œsophagus, tapering off upwards to reach the level of the bifurcation of the trachea. There seemed to be a slight constriction at the upper part.

Rent in the Œsophagus.

A male, aged sixty-two, suffering from acute mania, showed at the *post mortem* examination a rent in the left side of his œsophagus, with escape of gastric juice, a pint of gastric contents being present in the left pleura and leading to partial digestion of the mediastinal tissues and the lower lobes of each lung. There seemed no evidence of *post mortem* digestion of the œsophagus having caused the rent. There were some cardiac hypertrophy and a degenerated hydatid cyst in the liver. His death was attributed to rupture of the œsophagus, though there seemed no reason why this had occurred.

Post Mortem Digestion of the Œsophagus.

Post mortem digestion of the œsophagus was noted in two cases, the contents from the stomach having escaped into the pleura and led to partial digestion of the lung tissue *et cetera*.

THE SURGICAL TREATMENT OF DISLOCATED MENISCUS OF THE KNEE JOINT.

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Rupture or dislocation of the medial meniscus is the commonest lesion of the knee joint. The lateral

meniscus gives trouble, but less commonly. In my series of 160 patients with lesions referable to the menisci seen in private practice, the medial meniscus was affected in 146 and the lateral meniscus in 14. The only effective treatment is removal of the damaged meniscus.

It is surprising how often patients say they have been advised by their medical attendant not to have the knee opened. The risk, in the patient's mind, is that the knee will become stiff, presumably from sepsis. But the fear of sepsis is a relic of pre-Listerian times. One otherwise well-informed medical man asked me if I was not afraid to operate in a certain instance, because he had seen them go wrong in the best hands. Sepsis may result from infection if the joint is exposed for an inordinate length of time during operation. The time occupied by operation should not exceed fifteen minutes. In most cases ten minutes suffice to do all that is necessary. In my series there was no sepsis or other complication following operation.

Points in Diagnosis.

Frequent locking, with inability to extend the knee fully, is usually a sign of a dislocated meniscus. A depression in the position usually occupied by the meniscus, or undue prominence of the meniscus when compared with that in the sound knee, is another indication that the cartilage is not in its normal position. Pain over the menisci also indicates pathological changes in it due to trauma. In conjunction with the preceding signs recurrent swelling is an important sign.

What is Necessary to be Done at Operation.

The incision is made parallel to the patella, 1·25 centimetres (half an inch) from it, for about 5·0 centimetres (two inches), with the centre on the position of the knee joint. The knee is flexed for easy access to the menisci. Following the late Robert Jones's advice, one knife is used to incise the skin and a different one to cut the capsule and synovial membrane. When the meniscus is exposed it is lifted on a blunt strabismus hook and examined for lacerations, or failing lacerations, for fraying or crenation of the edges on the concavity. These, if present, indicate that the meniscus has been nipped in movement of the joint and should be removed. Another sign of dislocation of the meniscus is a small erosion on the cartilage of the inferior surface of the condyle of the femur. The points stressed under "Diagnosis" should be kept in mind when a decision has to be made as to whether the meniscus should be removed or not. In the absence of signs of injury, a history of frequent locking and inability to straighten the knee indicates that the meniscus should be removed, provided no loose bodies other than those derived from the meniscus are present. The stitching of towels to the edge of the wound is a waste of time and exposes the wound unnecessarily. The cut with the knife carries in more germs than would come from the sterilized skin around the wound.

The removal of a meniscus does no permanent damage to a knee joint, as its place is quickly filled by tissue from the synovial membrane. Overgrowth of synovial tissue may lead to subsequent pain of the joint simulating a torn meniscus with swelling from excess of synovial fluid. This, however, passes off in a few days with no permanent ill-effects, if active movement is resumed immediately.

It is necessary to remove the anterior portion only of the meniscus in most cases; that is, as far as its attachment to the capsule at the lateral sides of the joint. If the meniscus is freely movable posteriorly it may be drawn forwards and removed, but if firmly attached it should be left alone.

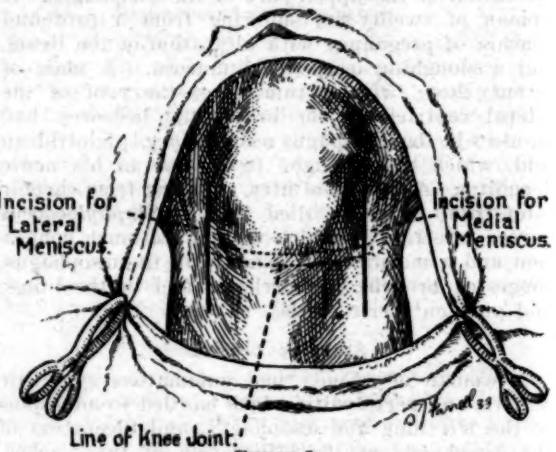

FIGURE I.
Showing sites for incision.

After-Treatment.

Much of the trouble following an operation is due to prolonged immobilization. Immobilization in plaster is to be shunned. Immediately after operation the knee is placed on a pillow and between sandbags, and is kept thus for three days. The patient moves the joint as soon as possible and gets out of bed on the fourth day. A splint made of folded newspaper and extending from the upper part of the thigh to below the calf is all that is necessary to prevent sudden flexion of the knee. The patient may walk without a splint at the end of the seventh day and leave hospital on this day. Massage may be necessary if the quadriceps muscle is wasted, but is a waste of time and money if not. Exercises for the quadriceps muscle are useful to restore power and tone; but electrical stimulation is valueless when active movements can be performed. The isolated wasting of the *vastus medialis* is a delusion, and depends on the presence of general wasting of the quadriceps. The *vastus medialis* normally overhangs the knee when in relaxation, whereas the *vastus lateralis* is not allowed to sag on account of the presence of the ileo-tibial tract on the outer side. The patient should be walking normally in fourteen days. The swelling from excessive synovial fluid which follows operation disappears with exercise and should disappear in this time.

Reports of Cases.

PNEUMOCOCCAL MENINGITIS.[1]

By RALPH WHISHAW, M.B., Ch.M. (Sydney),
Hobart.

J.T., AGED eighteen years, had been in good health for the past twelve years. Previous to that I had had a good deal of anxiety assisting him through an attack of double lobar pneumonia.

His first attack of meningitis came on with dramatic suddenness. On March 17, 1933, he went to bed with a headache, vomited several times during the night, and in the morning was found unconscious. He was restless and irritable, resenting examination, and appeared to be in pain, which was apparently due to a distended bladder. He had slight retraction of the head and taut neck muscles. Kernig's sign was present. His temperature was 38·3° C. (101° F.), his pulse rate 100 and his respiratory rate 24. He was unable to swallow. Thirty-five ounces of urine were drawn off, and this contained a large quantity of sugar and diacetic acid. After insulin had been injected and glucose had been given by the rectum the sugar disappeared from his urine. Lumbar puncture revealed dark blood-stained fluid under considerable pressure. The origin of the blood was not thought to be traumatic, as it was intimately mixed with the cerebro-spinal fluid and was dark in colour. The fluid yielded a pure culture of pneumococci (Dr. Walch).

No chemical tests were done on account of the large amount of blood present. Twenty-four hours after the puncture the patient regained consciousness and spoke rationally, complaining of headache and double vision. Examination at this stage revealed normal fundi and fields, active pupils, normal corneal sensations, a normal orbicularis, sixth nerve palsy (Dr. Hamilton). The paresis of his bladder remained, both knee jerks were absent, and Babinski's sign was present. X ray examination of the skull revealed no abnormality. The blood did not react to the Wassermann test. Forty-eight hours later he relapsed again into unconsciousness and became delirious and noisy. A second puncture yielded fluid that contained less blood and that was under less pressure. The following day he regained consciousness and made an uneventful recovery. At the end of three weeks he regained control of his bladder, and the knee jerks returned about the fifth week. Following the long period of catheterization, he developed an infection of his urinary tract, which cleared up at the end of three months. Four months after his illness an abscess developed in the natal cleft at the level of the junction between the sacrum and coccyx. This discharged pus in small quantities for two weeks and then completely healed.

The patient's second and last illness began on November 11, 1933, eight months after the first. During that time he had gained 18·9 kilograms (three stone) in weight, making his weight 101 kilograms (sixteen stone). He appeared to be in excellent health. Dr. Shugg saw him on November 17, 1933, and I am indebted to him for the following notes.

Three days previously he had abraded a small area of skin on the dorsum of his left foot, from a new pair of shoes. This had become infected and a localized cellulitis had developed, with some swelling. Later on the same day he had a rigor and complained of generalized pains, especially in the head and back of the neck. His temperature was 38·3° C. (101° F.), his pulse rate 100. A second rigor followed and the skin became moist. There was slight pain on moving the head, but otherwise examination revealed no abnormality.

On November 18, 1933, he was better and the temperature was normal, and only slight neck stiffness was present. On November 19, 1933, the pain in his head and neck became severe and a complete facial paresis appeared on the right side, with ptosis of the left lid. His pupils were widely dilated and fixed, and failed to react to light or accommodation, and generalized ophthalmoplegia was present. Neck rigidity was well marked. Kernig's sign was positive. No Babinski sign was present; the right great toe gave a flexor response and the response on the left side was equivocal. The patient was conscious, but occasionally irrational. He became worse during the day and semiconscious that night, with marked cerebral irritation.

When I saw him on the morning of November 20 he was quite unconscious, with wide staring eyes and stertorous respiration. There were marked neck rigidity and paresis of the bladder. Lumbar puncture revealed very turbid fluid, containing pus and pneumococci. He did not regain consciousness. On November 21, 1933, his temperature rose to 41·1° C. (106° F.), the urine became solid with albumin, and he died at 5 a.m., that is, on the fifth day.

Pathological Report.

I am indebted to Dr. J. H. B. Walch for the following pathological report.

On March 24, 1933, Dr. Whishaw brought me a sample of cerebro-spinal fluid. It contained a considerable amount of pus, with a large number of Gram-positive diplococci, which were cultivated and identified as pneumococci.

On November 20, eight months after bringing the first specimen, Dr. Whishaw brought me another sample of cerebro-spinal fluid from the same patient. Again it contained a considerable quantity of pus. The pus was of a flocculent nature, suggesting that the thick, purulent contents of an abscess had been mixed with the fluid, rather than a direct generalized infection of the meninges. Owing to the flocculent character of the pus, a cell count was impracticable. A column of the fluid, about 25 millimetres in length, rapidly deposited a layer of pus about a millimetre in thickness. A direct smear showed a very large number of Gram-positive diplococci, mostly intracellular, with many degenerating individuals. Capsules could be distinguished in some instances. The organisms were cultivated and shown to be soluble in bile. They were not agglutinated by pneumococcal sera of Type I, II or III, so it was concluded that they were Group IV pneumococci.

Next day Dr. Whishaw and I made a *post mortem* examination. We found a general cloudiness of the meninges over the vertex of the brain, with a few pockets of thick pus in the sulci. The under surface of the frontal lobes was clear, but immediately behind the optic chiasma we came on a large collection of thick yellow pus, which extended backwards round the base of the brain to the *cisterna cerebello-medullaris*. The fluid extruded from the spinal canal was thin and watery, with flocculles of pus, similar to that obtained by lumbar puncture, and quite different from the thick yellow pus in the *cisternæ subarachnoideales*. The floor of the cranium was carefully examined for a point of entry of the infection, but none was found. The tympanic antra were opened and found healthy. The cavernous and transverse sinuses were not involved in the infection, and the whole bony structure of the cranium seemed intact and healthy. Sectioning of the brain revealed no collections of pus in its substance.

The pericardium contained a little fluid and a small deposit of fibrin, but the heart muscle and endocardium seemed normal. The right pleura contained about a pint of fluid, coloured dull brown with hæmatin, and a considerable fibrinous deposit. The surface of the right lung was similarly coloured, but its interior seemed normal. The whole of the left lung was in a state of red hepatization. The abdominal organs showed no significant lesions.

[1] Read at a meeting of the Tasmanian Branch of the British Medical Association on December 12, 1933.

Post Scriptum.

Since this paper was read, Dr. W. L. Crowther has informed me that recently he treated a patient who suffered from meningitis and from whom pus containing pneumococci was obtained by lumbar puncture. Polyvalent serum was used and the patient recovered.

THE PERITONEUM AND INFECTION: A CASE OF APPENDICITIS.

By T. M. FURBER, M.B., Ch.M. (Sydney), F.R.A.C.S.,
Honorary Surgeon, Coast Hospital; Honorary Assistant Surgeon, Royal Prince Alfred Hospital, Sydney.

I SUPPOSE it started with our histology lectures, when we were taught that the serous membranes were very delicate structures, but it took me some years of surgery to realise that the peritoneum is not a liability, apt to kill by becoming inflamed, but is a very secure asset, able, if not overwhelmed by the sudden rupture of a hollow viscus, to deal with most of the infections it meets in its day's work and to make good the rough handling and leaky stitching with which it is so often insulted.

The formation of a retroperitoneal abscess as a result of appendicitis must in these days of enthusiastic operators be rather uncommon, so that the following case seems worthy of a brief note, if only to stress again the intrepidity with which the peritoneum deals with violators of its sanctity.

A man, R.H., aged forty-two years, had, fourteen years ago, three attacks of pain in the right side of the lower part of the abdomen, accompanied by constipation, dizziness and disinclination to work. The attacks came at about four months' intervals, lasted three or four days, and were followed by local soreness. Eleven years ago he felt a sudden burning pain in the right side of the lower part of the abdomen, which made him draw his right leg up and which gave his medical attendant some difficulty to diagnose, till it was recognized by Dr. Edgar Stephen, who was called in after a week, that the condition was one of appendicitis which was going on to form an abscess. The patient was admitted to the Western Suburbs Hospital and the abscess was drained, and in due course healed. It recurred three times, the last one being the occasion on which Dr. Clive Robinson asked me to see him, with a view to removing the offender when the abscess had settled down. The surgeon who had attended the patient in the previous two illnesses had advised against any further surgery.

On October 17, 1933, I opened what was apparently a retroperitoneal abscess extending from the vertebral column medially to a vertical line from the anterior superior spine laterally, and from the ribs above to half way down the iliac fossa. It settled down, and on December 5, 1933, we opened him with a view to removing the cause of the trouble.

A Battle's incision was chosen, as it seemed more likely to give more room for manœuvring, and it gave quite good access to an appendix which was easily separated from the medial aspect of the meso-caecum, starting at the caecal end. The sting, however, was in the tail, and when the distal end was carefully and with difficulty separated, it was possible to reconstruct the pathology as follows. Incidentally one was struck by the notable absence of adhesions.

Apparently at the first attack the appendix had ruptured about a quarter of an inch from the tip, and the intraperitoneal abscess which formed was drained in the usual place. When the abscess walls contracted and the adhesions were resorbed, the edges of the perforation alone remained adherent to the posterior peritoneum. In this there was a gap leading to a small extraperitoneal cavity, which just took the tip of my forefinger and was situated a little above the brim of the pelvis, and into which the mucous membrane of the appendix pouted like a miniature colostomy.

The peritoneum had not only cleared up the mess from the previous fracas, but had neatly arranged that whenever another attack of appendicitis came, the appendix would drain outside the peritoneum, probably saying to itself, with a sigh of relief, as so many of us selfishly do, "Thank heavens I'm all right, let the other fellow look out for himself"; although, when you come to think of it, it would probably be better for the patient to have an extraperitoneal rather than an intraperitoneal abscess.

I saw the patient on February 17, 1934, and all wounds were soundly healed and he had resumed work without any untoward symptoms.

PLEURISY WITH EFFUSION.

By G. M. HAYDON, M.B., B.S. (Melbourne),
Maffra, Victoria.

M.M., AGED twenty-three years, consulted me first on July 28, 1933, complaining of "flu" and pains in the chest. Examination revealed a not very sick young man with a faint friction rub at the left base posteriorly.

On July 30, 1933, the friction rub was still present, but disappeared next day, owing to the accumulation of fluid. His chest was tapped on August 3, 1933, and 450 cubic centimetres (fifteen ounces) of clear fluid were drawn off with Potain's aspirator. On August 5, 1933, 1,200 cubic centimetres (two pints) of clear fluid were drawn off. This did not seem to be all the fluid, but as the patient was slightly restless no more was drawn off just then.

On August 9, 1933, 600 cubic centimetres (one pint) were drawn off. On August 10, 1933, 1,200 cubic centimetres (two pints) were drawn off. On August 15, 1933, 1,800 cubic centimetres (three pints) were removed.

On August 21, 1933, 450 cubic centimetres (fifteen ounces) of slightly thicker fluid were drawn off, so that altogether 5·7 litres (nine and a half pints) of fluid were drawn off.

His temperature throughout this ranged to 38·9° C. (102° F.) and at no time did he appear dangerously ill. He suffered from an attack of synovitis of the extensor tendons of the right wrist on January 2, 1934, when attempting to lend a hand on the farm.

He has diminished breath sounds at his left base now, but otherwise appears relatively fit. I have advised him to take extra care of himself, thinking of the possibility of latent tuberculosis.

The case is reported to show the large amount of fluid aspirated.

PRIMARY LARYNGEAL DIPHTHERIA IN AN ADULT.

By J. STEIGRAD, M.B., Ch.M. (Sydney), F.R.A.C.S.,
Honorary Relieving Assistant Surgeon, Royal Alexandra Hospital for Children, Sydney.

THE record set out below is considered worthy of reporting owing to the rarity of the condition and the ease with which the diagnosis may be missed. Laryngeal diphtheria in adults is uncommon, and primary laryngeal diphtheria in adults is very uncommon. In fact, statistics at the Royal Alexandra Hospital for Children show that even in children of all ages secondary laryngeal diphtheria, that is, laryngeal diphtheria occurring consequent upon faucial, nasal or naso-faucial diphtheria, is eight or nine times more common than primary laryngeal diphtheria.

The patient, a third-year nurse, aged twenty-one years, on the staff of the Royal Alexandra Hospital for Children, was first seen by me when I was Chief Resident Medical Officer of that hospital, on January 3, 1934. She had failed to react to the Schick test when it was applied some twelve months previously, and she was at the time nursing patients suffering from diphtheria in the isolation block of the hospital. She complained of an irritating cough and hoarseness of voice for one day. She did not look sick or toxic, there was no stridor or dyspnœa, and her

chest was quite clear. The fauces were innocent of even injection, and she was thought to have the laryngitis and tracheitis so commonly seen among the nurses of large hospitals. Bacteriological examination after a throat swabbing failed to reveal the Klebs-Löffler bacillus. Seen the next day, she was completely aphonic, and the posterior pharyngeal mucosa and the posterior tonsillar pillars were slightly injected. Another throat swabbing was done on this day and still diphtheria bacilli were not found. The following day, however, she had small patches of membrane on the posterior parts of the tonsils and on the posterior pharyngeal wall. Examination of an immediate smear of this membrane revealed the presence of the diphtheria bacillus. Diphtheria antitoxin in a dose of 30,000 units was administered intramuscularly, and later in the day, on several occasions, the patient coughed up membranous casts of the larynx and the trachea. Further antitoxin was given in doses of 20,000 units the following day and 10,000 units the third day, making a total of 60,000 units in all. The progress was satisfactory and the patient is now convalescing.

Reference to Dr. F. V. G. Scholes, of Melbourne, has brought the interesting information that in his vast experience of diphtheria, laryngeal diphtheria in adults is rare (less than 2% of all cases of laryngeal diphtheria), and that adult primary laryngeal diphtheria is very rare. He also pointed out the high mortality rate, due to lateness of diagnosis and consequently of treatment.

Reviews.

ORGANIC CHEMISTRY AND BIOCHEMISTRY.

THERE is room for divergence of opinion as to the best method of treating organic chemistry when it is intended as a preliminary to the study of biochemistry, as, for instance, in the instruction of medical students. There can be, however, no disagreement on certain points, namely, that the subject matter must be accurate and clearly expressed, fundamental methods must be adequately dealt with, while unnecessary technical details may be omitted, that a proper balance between the important and the unimportant must be maintained, and finally, that older, purely academic methods must give place to those methods more in the forefront of the modern science. The organic section of R. H. A. Plimmer's work on organic chemistry and biochemistry fails to some extent in all these directions.

There are several unfortunate inaccuracies in both fact and expression. On page 18 is a lamentable example: "The elementary composition, or detection of the elements, precedes the quantitative composition." On page 80 we are informed that alcohol yields acetaldehyde by "dehydration" in the presence of silver as catalyst. These are only typical of the looseness of expression and inaccuracy of many parts of the book.

A lack of care in the selection of topics is often apparent. Space is occupied by mentioning and giving the equation for the preparation of dichloracetic acid, while reference to such an important class of substances as the Grignard reagents is excluded, presumably on the grounds of lack of space. Again, all mention of modern quantitative micro-methods is omitted from the chapter on the estimation of the elements, and many modern preparative methods, such as the use of thionyl chloride for the preparation of acyl chlorides and the formaldehyde-ammonium chloride method for methylammonium chloride, are passed over.

According to the preface, the book was intended originally as a guide to practical work. In this respect it is open to the criticism that there is far too much test tube and colour reaction work. The instructional value of innumerable tests, the results of which are known beforehand, is very small, and unless the chemical reactions involved in such tests are emphasized, their introduction into a practical course is an encouragement of perfunctory work on the part of the student.

We are surprised to find that such an important biochemical process as the synthesis of carbohydrates by plants is passed over in a few lines, while, in striking contrast, the correct method of figuring the pyranose structure of glucose, which is, after all, an insignificant and purely academic matter, occupies considerable space. As there are many elementary text books of organic chemistry suitable for medical students, there seems little room for a work of the character of this one.

[1] "Organic and Bio-Chemistry", by R. H. A. Plimmer, D.Sc.; Fifth Edition; 1933. London: Longmans, Green and Company. Royal 8vo., pp. 634, with illustrations. Price: 21s. net.

THE ART OF THE PHYSICIAN.

THERE are books—legions of them—which do honour to the intellect of their purchasers. Such, for instance, are the lighter writings of Osler, the poems of Keats, the works of Stephen Paget, the "Religio Medici", the "Horae Subsecivae", the mingled tears and laughter of the "Vicar of Wakefield"; and all these the works of doctors. And now has come a volume more than worthy to stand beside these other children of our colleagues' brains, a fit companion of our hours of solitude and contemplation. Dr. A. G. Gibson has expanded, in a philosophical vein, the tattered fragment of John Locke's manuscript, "De Arte Medica".[1] The thing is neither more nor less than an attempt to portray the physician as he should be and, as we hope, he most usually is. A quotation or two will indicate the general tenor and scope of the book.

> A large proportion of the ailments met with in practice are trivial and unimportant in their relation to life. An attack of mumps or chicken-pox in a healthy child is of this order; and so are many skin diseases, but some, such as ringworm of the head in a child, or acne of the face in a girl, assume a prognostic importance from their social aspect . . .
>
> It is not right to deny a man stricken with mortal disease any food or remedy he may desire to try, even though it is clear it will do him no good . . . It will do him no harm to try these things, and it may be nature's way of telling him his days are numbered.
>
> In all grades of practice there is work for which no payment is possible. Poorer patients cannot afford to pay adequately for what is done for them, and there are many others whose burdens ought to be lessened by the charge of smaller fees during a period of stress . . .

A good man—a great man! And he can—he certainly can—write English prose.

ANOTHER VENEREAL DISEASE.

"A SIXTH VENEREAL DISEASE", by Dr. Hugh Stannus Stannus, is a survey of contributions by various authors of conditions known as climatic bubo, *lymphogranuloma inguinale*, esthiomène, chronic ulcer and elephantiasis of the genito-ano-rectal region, and inflammatory stricture of the rectum.[2]

The author asserts that evidence has recently been adduced to show that the group of conditions mentioned is due to infection with the same virus, and that one more

[1] "The Physician's Art: An Attempt to Expand John Locke's Fragment de Arte Medica", by A. G. Gibson; 1933. Oxford: The Clarendon Press. Crown 8vo., pp. 237. Price: 7s. 6d. net.

[2] "A Sixth Venereal Disease", by H. S. Stannus, M.D., Ph.D., F.R.C.P., M.R.C.S., D.T.M. & H.; 1933. London: Baillière, Tindall and Cox. Demy 8vo., pp. 282, with illustrations. Price: 12s. 6d. net.

disease should be added to the list of venereal disabilities. In support of his contention, he appends a list of 933 titled articles by various authors covering a century of observation. The literature quoted is presented in a methodical and terse manner and is linked by personal observations and criticism, the result being a most readable book.

One wishes that an alternative title had been provided for the publication, so that an attempt might have been made to guide opinion towards a suitable name for the disease. An inclusive name has not yet been suggested, though climatic bubo and *lymphogranuloma inguinale* are together spoken of as poradenitis—a term in use by Continental writers and suggested by Fiessinger in 1925. The author favours the name poradenitis in preference to *lymphopathia venericum*, suggested by Salzberger and Wise in 1932, although he thinks the latter has many points in its favour.

The book is well bound, the printing is clear and easily read, and the index is carefully classified and is a good guide to the contents. There are sixteen well printed illustrations on nine plates.

The volume should especially appeal to those interested in venereal diseases, as well as to dermatologists and gynæcologists, and can be recommended as worthy of a place among medical books of reference.

DISEASES OF CHILDREN.

IN its latest edition, the well known book on diseases of infants and children, by H. D. Chapin and L. T. Royster, appears with the title altered to "Pediatrics", in keeping with modern usage and with the subject matter of the book, in which several chapters are devoted to the development, growth, nutrition and metabolism of the healthy child.[1] One of these chapters deals with "the appraisal of the child", which denotes an attempt to ascertain by thorough physical examination and without reference to any fixed standard of normality, the status of the individual child and its ability to adapt itself to the ordinary activities of childhood.

Medical knowledge has during recent years advanced in so many directions that it is often difficult for the general practitioner, or even the specialist, to keep up with it. The authors have shown commendable discrimination in their selection of new material for the latest edition of their book. The biochemical basis of disease, which has of late occupied a prominent place in medical research, has received due attention in the discussion of such conditions as acidosis, rickets and tetany; attention is drawn to the important part played by the latter in the ætiology of infantile convulsions. The authors stress the distinction between ketosis, which results from incomplete oxidiation of fat and which in itself may call for no special treatment, and the more serious condition of acidosis, which results from an alteration in the acid-base ratio, and which in children is more readily precipitated on account of the frequent occurrence of conditions associated with anhydræmia.

There is much new material in the chapter on anæmia; the classification of the anæmias of childhood into deficiency and hæmolytic anæmias, however, would seem to be preferable to a classification "by convention and tradition" into primary and secondary groups.

A special article on nephrosis has been introduced; also in the section on nutritional diseases particular mention is made of the success in some quarters attending the treatment of diabetes with high carbohydrate diets.

The reference to the serum treatment in poliomyelitis as having been "used with supposed success" is disappointing, as many promising results have been associated with its use in the early stages of the disease, and although controls are lacking in these instances, the advantages must surely outweigh any disadvantages in such a distressing disease.

The illustrations are good: but it is a pity that some which appeared in the previous edition, particularly in the chapter on infectious diseases, have been omitted.

This book, in which the subject of pædiatrics is presented in a comprehensive and compact manner, can be recommended with confidence to the student and general practitioner, for whom it is intended.

[1] "Pediatrics", by H. D. Chapin, M.A., M.D., and L. T. Royster, M.D.; Seventh Edition; 1933. London: Baillière, Tindall and Cox. Royal 8vo., pp. 791, with illustrations. Price: 35s. net.

PATHOLOGY.

IT is less than ten years since Robert Muir, of Glasgow, first published his "Text Book of Pathology". The third edition keeps the book in its well-earned place, that of one of the very best of the single volume works on the subject.[1] It is of great interest to read this new edition with the previous edition at hand, for not only is it readily possible to verify the care and thoroughness with which the revision has been made, but it is striking to observe how very important the advances in pathology are in the realm of practical medicine. This is also a tribute to the way in which Professor Muir handles his material; he is an admirable exponent of the pathology of the living, and never loses contact with the actual problems of disease. The book is about eighty pages longer, but by selecting a paper of appropriate weight the new volume is apparently no larger. It is, as before, profusely and well illustrated, and in adding to the various sections as required by the new work done in the last four years, the author has maintained balance of the book. A good deal has been rewritten, and the style, one of the admirable features of the book, is just as lucid and pleasant as before.

The writer does not hesitate to show how completely certain of the advances in pathology have altered his personal views, as, for example, in the case of pernicious anæmia, where he previously was a strong supporter of the theories of William Hunter. He appears to have definite doubts as to the validity of the suggestion of Witts that pernicious anæmia is not of the hæmolytic order. The section on coronary disease is another excellent example of the clear and full account of recent work in its bearing upon the problems of cardiac disease. In turning to the section on renal disease it is naturally interesting to see whether the author has been influenced by the recent elaborate classifications of nephritis. But while adequate references are given, he has not permitted the simple presentation of the subject to suffer. A vexed topic like nephrosis receives a brief and uncomplicated treatment, as is proper in a work of this dimension. The pathology of œdema has been more adequately dealt with in this edition and, as before, is aptly placed in the section on disturbances of the circulation. Endocrine advances, one would hardly expect to see described very fully, considering the vast amount of new literature appearing, but the additions are adequate and contain the more important and less speculative details. The chapter on tumours has also been expanded, and the essential additions to knowledge have been incorporated. It is not necessary to refer to further amplifications and alterations in this reliable work. It is sufficient to state that it is well up to date, eminently readable, and suitable not only for the undergraduate student, but also for the practitioner of medicine. Surely anyone with a serious practical interest in clinical medicine and surgery will find on turning up a few references in these pages that he will continue to read on with both pleasure and profit.

[1] "Text-Book of Pathology", by R. Muir, M.A., M.D., Sc.D., LL.D., F.R.S.; Third Edition; 1933. London: Edward Arnold and Company. Royal 8vo., pp. 964, with illustrations. Price: 35s. net.

The Medical Journal of Australia

SATURDAY, MAY 5, 1934.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

CHRONIC NEPHRITIS IN QUEENSLAND.

THE prevalence of chronic nephritis in Queensland is well known to readers of this journal. The mortality from acute nephritis is higher in Queensland than in any other State in Australia. This statement holds good for all age groups, and the mortality is particularly high during the third and fourth decades. The mortality from chronic nephritis in the first, second, third and fourth decades is much higher in Queensland than in any other State. In the third decade it is over five times that of the next highest rate. On previous occasions we have laid stress on the obvious fact that some factor is at work in Queensland that does not operate in other States. Medical practitioners cannot fail to be aware of the pioneer work of Dr. J. Lockhart Gibson and Dr. A. Jefferis Turner and of the subsequent investigations of Dr. L. J. J. Nye on the causation of lead poisoning in Queensland children by the absorption of dried and powdered lead paint. Nor can they be ignorant of the view, so ably put forward in recent years by Dr. Nye, that lead from this source is the cause of chronic nephritis.

In the past we deplored the fact that the authorities in Queensland ignored the prevalence of chronic nephritis in the State and that they did not appear to realize that plumbism was unduly prevalent among its children. This official apathy was terminated when the Federal Government, at the instance of the Queensland Branch of the British Medical Association, agreed to investigate the question. The report of this investigation was not published, but a summary of the findings was published in *Health* in December, 1932. As was pointed out by Dr. Nye in this journal in the issue of August 19, 1933, the report upheld the contention that kidney disease is unusually prevalent in Queensland, and agreed that lead was in some way concerned in the renal syndrome. It was at this stage that the Queensland Government took official notice of the problem. In May, 1933, the Premier of Queensland communicated with the Secretary of the Walter and Eliza Hall Institute of Research in Pathology and Medicine, Melbourne, and requested the Institute to review for the Queensland Government the whole of the evidence on the question of chronic nephritis in Queensland. The Institute was asked specifically to state: (i) Whether there was sufficient evidence incriminating lead paint as a causative factor in chronic nephritis in Queensland. (ii) If not, what further evidence was necessary in order to arrive at final conclusions with a view to preventing the appalling mortality from the disease. The Institute acceded to the request of the Premier and entrusted the work to Dr. Keith Fairley. A copy of Dr. Fairley's report has been received from the Premier's Department, Queensland, and is published *in extenso* in this issue.

The answer given by Dr. Fairley to the questions put forward by the Queensland Government is quite definite. He states that there is sufficient evidence to incriminate lead paint as a causative factor in chronic nephritis in Queensland. He also expresses the opinion that legislation regarding lead paint should be directed against the use of this paint on any article available to children. He states quite rightly that partial prohibition is likely to be ineffective and to give rise to a false sense of

security; to be effective, prohibition must be complete and unconditional. With these conclusions we agree entirely. We also agree that what Dr. Fairley calls the veranda theory has not been scientifically proved. As he points out, scientific proof of this theory would necessitate the unjustifiable exposure of large numbers of children to what is believed to be grave risk of permanent damage. Everyone will be prepared to admit that further intensive study, particularly of basophilic degeneration in large groups of children, would shed more light on the subject. But even study of this kind might not produce irrefutable scientific proof. If the use of all lead paint in certain places were prohibited, time would show whether lead was responsible.

As a final conclusion of the matter, it may be stated that the duty of the Queensland Government is now clear. Having received a definite reply from an authority of its own choosing, it must without delay take the next step and prohibit the use of lead paint in certain situations. If it shirks this duty, it stultifies itself and shows an utter disregard for the health of its people.

Current Comment.

THE FATTY HEART.

A DIAGNOSIS of "fatty heart" was once made confidently by clinicians of an earlier day. This term really included the two diverse pathological states of fatty infiltration, in which the fat penetrated from an unduly profuse adipose epicardial envelope amongst the muscle fibres, and fatty degeneration, in which lipoid deposits actually formed within the muscle cells. Now the pendulum has swung in the opposite direction, and physicians hesitate to visualize the adipose heart, in spite of the knowledge that such changes do occur. Harry L. Smith and Fredrick A. Willius, from the Cardiological Department of the Mayo Clinic, have made a clinical and pathological study of this subject of adiposity of the heart, and present an analysis of 136 cases.[1] They point out at the outset that the current view is that intracellular degeneration is due either to toxæmia or to disturbance of cellular nutrition. The former cause is, of course, distinct: where obvious infective or otherwise toxic disease exists some cellular lipoid changes may be expected. The latter is of greater importance, for it will be seen that infiltration of the heart with fat will cause a disturbance of nutrition in the muscle cells; thus the variety of fatty heart most clinically important is that occurring in obese patients. Amongst these patients at least the two pathological varieties of fatty heart may be included under the one heading of adiposity of the heart.

Smith and Willius have studied autopsy material in 136 cases of persons who were selected for observation simply because they were obese, the degree of increase in body weight being never less than 13% above the standard normal for age, sex and height. The females in this series were more than twice the number of the males. The method employed was to make a quantitative assay of the fat in the heart muscle, to carry out histological studies and to link up the findings with the clinical state observed before death. It is of interest to note that the heart was found normally to be 0·43% of the body weight in men and 0·4% in women, and the method of computing the heart weight was found to be correct within 8% to 10%. In 52 out of the 136 cases obesity existed without evidence of any definite heart disease, but it was noted that the weight of the heart in all cases fell below the normal percentage of body weight, though the actual weights were increased. A small but interesting group, nine cases only, was that including cases of cardiac insufficiency in which no heart disease could be demonstrated. Here again the hearts were actually heavier than usual, but still below the normal percentage of body weight; this must surely be significant, for several of these patients died of congestive failure. But in the hypertensive series a different state of affairs was found. Here, as might be expected, there was a considerable increase, not only in the average actual weight of the heart, but also in its percentage of body weight. The fatty hypertensive hearts were of a weight greater than could be explained by the increase in body weight alone. The last group studied was that of cases of obese patients with known cardiac disease.

Certain clinical features of interest were noticed. Hypertension occurred in 44% of the patients, and adding to these the number of cases of other circulatory disease the total percentage of cardiovascular affections in the obese was 62. One interesting fact is that no less than 19% succumbed to pulmonary embolism. As the authors remark, one remembers in this connexion that obese patients are "poor surgical risks".

With regard to the types of fatty affections of the heart, Smith and Willius believe that a deposit of fatty globules within the muscle fibres affects the function of the heart but little. The infiltrative type here studied they do believe of importance. They do not consider that it constitutes a great danger of cardiac insufficiency *per se*, but that it adds a dangerous burden to other circulatory dis-

[1] *Archives of Internal Medicine*, December, 1933.

turbances, such as hypertension, coronary or endocardial disease. The separation of the muscle fibres by excessive fat is apparently the chief factor that constitutes this additional burden. The final conclusions arrived at are that occasionally the large heart of the obese tends to be relatively below the average for body weight, that it may sometimes fail (presumably because of its inadequacy for the task in hand) irrespective of other heart disease, and lastly that obesity, both general and cardiac, is a contributory factor in producing cardiac failure. To this may be added the reminder that obesity carries with it a potential vascular vicious circle. The more inadequate the heart becomes to deal with the rising body weight and the more the body weight increases, the less able and the less inclined is the patient to lead an active life. This in its turn augments the existing defect in tissue oxidation, and more fat is laid down. A recent writer has defended the obese, claiming that "we would rather die in full possession of our faculties in our middle age than totter about in scrawny-legged misery for a few extra years". This is a matter of opinion; perhaps there are still those who would preserve their hearts with their years, even though they lose their curves.

IMMUNITY AND MODERN RESEARCH.

To anyone who spends a little time in looking back over the progress of knowledge in the vast subject of immunity, it must be obvious that, despite notable advances on the experimental side, the bedside harvest has not been yet fully reaped. The introduction of antiserum into clinical medicine, the use of vaccines, particularly in prophylaxis, and the elaboration of the laboratory tests now in daily use altered the whole aspect of medical work. But since those days the clinical bearings of immunology have been less evident. Diagnosis and prophylaxis have perhaps received most benefit, for even the question of the value of vaccines in treatment cannot be considered to be settled. It is at periods such as these that the "ultra-clinician" is perhaps disposed to overlook or even to view with a touch of good-natured tolerance the results of workers in the realm of pure science. But it must not be forgotten that an eminent man dismissed Hertzian waves as of little practical importance, but a short time before the transformation of the world by Marconi. Similarly it may be that we are on the eve of most important practical applications of theoretical work to everyday medicine.

A most valuable collection of the available knowledge concerning the chemistry of antigens and antibodies has recently been made by J. R. Marrack at the instance of the Medical Research Council.[1] This publication is, of course, interesting in detail only to the physical chemist working in the immunological field, but certain parts of the subject should attract the attention of all who are in any way concerned in the cure and prevention of infective disease. The first part of the monograph deals with the physico-chemical considerations involved, and is followed by discussions on the nature of antibodies and the specificity of antigens. Hitherto it has been assumed that antibodies are of protein nature, and modern work seems to indicate the very close relationship of antibodies with proteins; at least no attempts to prepare protein-free antibodies have met with success. Similarly the complex formed by the union of antigen and antibody contains a definite amount of globulin derived from the antiserum, and experiment seems to show that antibodies consist of a modified serumglobulin. But when we turn to the question of the antigen, its physical character and its specificity, other considerations arise. No longer can we confine our studies on antigens to protein substances. Clinical contacts are here made by the allergist, who is aware of the part played by non-protein substances, though, of course, here the problem is not purely one of antigen and antibody. But there are most interesting possibilities in connexion with compounds such as the polysaccharides and the lipins. Numbers of polysaccharides are known which display a specific reaction with corresponding antisera. Of these one of the most important is found in the capsule of the Type III pneumococcus, which is known to be resistant to ordinary immunological experiment and to be the cause of a serious and often fatal form of lobar pneumonia. Immune reactions have also been demonstrated with isolated lipins, so it will be seen that many interesting developments may be expected, for fat metabolism in all its bearings appears to be linked up with many of the important degenerative and toxic changes that go on in the human body.

In discussing the antigen-antibody reaction, Marrack points out that, although non-protein substances may react with antisera, they are not sufficient for immunization, which requires protein, or at least some large-moleculed substance. For example, some successful efforts appear to have been made to immunize animals to the Type III pneumococcus by utilizing its specific polysaccharide coupled with protein or other adsorbed substance.

The various hypotheses of the basis of immunity are also briefly touched upon, and it is interesting to find that the most coherent basis for speculation and experiment is still given by the Ehrlich side-chain theory, on which most medical practitioners cut their immunological teeth as students. This brief account does not do even scant justice to a brochure which summarizes modern work in a highly technical field, but it may help to draw attention to the vast bulk of research work that is being done and to stimulate interest and awaken the hope that ere long every practising physician may be able to bring to the bedside methods that are the fruits of these labours.

[1] J. R. Marrack: "The Chemistry of Antigens and Antibodies", Medical Research Council of the Privy Council, Special Report Series, Number 194; 1934. London: His Majesty's Stationery Office. Pp. 134. Price: 2s. 6d. net.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Hole in the Macula.

J. W. CRAWFORD (*Archives of Ophthalmology*, December, 1933) has had the good fortune to observe, step by step, the actual development of a hole in the macula in a previously healthy retina. The patient, a woman of sixty-two, was seen in October, 1930, for a refractive error. Both macular areas were normal and both fields. In August, 1931, her left vision had failed slightly and in the macular region was seen a very small area of chorioretinitis. In July, 1932, the vision of the left eye was reduced to 0·1. Examination revealed a typical hole in the macula. The macular hole is generally described as a sharply circumscribed circular or oval punched-out area, corresponding closely in size to the macular reflex seen in the normal eye. It is depressed below the level of the retina, is reddish in colour and its floor studded with tiny white or yellow spots. Surrounding the hole may be seen a corona, paler in tint. It was first described by Knapp in 1869. Many of the reported cases are of traumatic origin and Kuhnt first recognized the non-traumatic type, following retinal vascular changes and degeneration.

Acute Inflammations of the Orbit in Children.

R. C. GAMBLE (*Archives of Ophthalmology*, October, 1933) emphasizes the importance of the ætiology in cases of orbital abscess in determining the correct treatment. Speaking generally the causes usually assigned are direct infection from wounds, inflammation from adjacent parts and embolism in scarlet fever, meningitis and influenza. The treatment usually advised is early incision over the supposed site of the abscess and drainage. Experience with the disease in 25 children has convinced the author that there are important differences in the clinical course in children and also differences in the proper treatment. At birth the frontal sinuses are not developed and are not large enough to be a factor until the seventh year. But there is an intimate relation between the orbit and the ethmoid sinus and to less extent the antrum, and the majority of the author's cases were in this group. Usually the swelling of the upper lid occurs suddenly and there is almost always a greater discharge from the nostril on the same side. Swelling of the lower lid follows a day or two later. Quite often this is as far as the process goes and after several days the inflammation subsides or the eyeball becomes proptosed and perhaps pushed towards the temporal side. Fixation of the globe and chemosis are signs that pus has formed. The cornea needs watching against the dangers of abrasion and exposure. An antiseptic ointment should be used. Subcutaneous induration above or below the internal canthal ligament may indicate where to open the abscess. The author believes that the process is really a subperiosteal abscess which elevates the periosteum, causing the proptosis, and which then burrows slowly forwards to burst on the skin near the canthal ligament. It is more serious when the abscess points under the conjunctiva. Radical intranasal operation on the ethmoid sinus, though it may be and has been successful, should be avoided on account of the danger of meningitis and metastasis of septic foci. The author advises tampons of epinephrine and silver protein in the nose, nasal suction and cold applications for the eye in the early, and hot in the later stages. If an abscess points it is opened with a Graefe knife, no pressure is exerted nor drain used. If then there is no improvement, an incision should be made as for an operation on the tear sac and the periosteum elevated backwards until pus is found. The cellular contents of the orbit should not be entered and incisions in the conjunctival fornix where the drainage is not good should not be made.

"Nupercaine" in Ophthalmology.

M. K. BOCHNER (*Archives of Ophthalmology*, December, 1933) gives a short summary of his experience with "Nupercaine" during the last three years. "Nupercaine" is a derivative of quinoline, readily soluble, and is best prepared by dissolving it in normal salt solution and kept in alkali-free glass containers. The addition of four grammes of boric acid to 100 cubic centimetres of solution (20 grains to an ounce) preserves it from the growth of fungi. Its anæsthetic effect is estimated to be ten times that of cocaine. It is said to be four times as toxic as cocaine, but since the dilution is one-tenth that of cocaine, it is relatively less toxic. After some previous trials the author uses it in 0·5% solution for consulting room work and 1% in surgery. For infiltration he employs a 1 in 1,500 solution (never stronger than 1 in 1,000) with 10 milligrammes of epinephrine to every 100 cubic centimetres. It does not increase ocular tension and has no influence on the pupil. The author considers it a most satisfactory anæsthetic.

Mydriatic Glaucoma.

S. V. ABRAHAM (*Archives of Ophthalmology*, December, 1933) is impressed with the paucity of references to mydriatic glaucoma in the literature of acute glaucoma. He has found only 26 cases on record and these include only those produced after the use of mydriatics in patients previously free from clinical signs of glaucoma. *Questionnaires* were sent to 207 ophthalmologists. From 82 who replied a total of 29 cases were reported. The condition occurred more frequently after the use of the longer lasting mydriatics and occurred most frequently in females. The author's inquiries also showed that the use of a myotic after mydriasis reduces the incidence. The danger in children has no basis, and this applies with almost equal force to all persons under thirty years of age. Among patients over thirty the incidence is 0·0217%.

Mooren's Ulcer Cured by Keratotomy.

S. R. GIFFORD (*Archives of Ophthalmology*, December, 1933) describes the inevitable progress of the advancing edge of the corneal ulcer until the whole cornea is destroyed. The hopeless prognosis prompted the author to try the effects of a delimiting keratotomy. Cauterization has proved useless; a conjunctival flap has also failed, though Kreiker reported success in three cases in which a flap was drawn from above to cover the cornea completely. In the first case, that of a woman of fifty-two, keratotomy was performed, the cut being four millimetres long and just in advance of the central border. The wound was reopened seven times during the following eleven days. Five months later the eye was quiet with vision of $^{20}/_{30}$. In the second case, that of a man of sixty-five, the incision was kept open twice daily for ten days. The ulcer healed from the periphery and never advanced beyond the incision. Vision $^{20}/_{30}$. The third patient was a woman of thirty. In this case after cauterization with trichloracetic acid failed, a conjunctival flap was drawn beyond the advancing border after the corneal surface was treated with the acid. The result was good.

OTO-RHINO-LARYNGOLOGY.

Partial Thoracic Stomach with Short Œsophagus.

J. P. MONKHOUSE AND S. K. MONTGOMERY (*The Journal of Laryngology and Otology*, November, 1933), summarizing their paper, report seven cases of partial thoracic stomach with short œsophagus. In six the diagnosis was confirmed by œsophagoscopy, and in one it rested on X ray evidence alone. These cases fall into two groups, those with and those without dysphagia. In both types there is pain resembling the flatulent dyspepsia of cholecystitis. The dysphagia is not steadily progressive, as in carcinoma, but is intermittent, and for some time, often years, is not severe. It is due to the presence of an ulcerated stricture. Hæmatemesis may occur in both groups. The diagnosis rests on X ray and endoscopic examination. In the former, unless the barium is given in the manner described, the lesion is not seen, and it is essential that the passage of the opaque material should be followed on

the screen in order to differentiate the condition from a paraœsophageal hernia. In cases with dysphagia stricture is revealed by œsophagoscopy, possibly with visible ulceration, and mucous membrane removed from this level is found to be gastric in type. In the second group no stricture is seen; but a dilatation may be observed at a level that is certainly above the diaphragm and from which gastric mucosa is obtained. Dilatation will relieve the symptoms in the obstructive type; patients without dysphagia receive some benefit from prolonged medical treatment, but do not respond well. The pathology is discussed, but owing to the lack of *post mortem* material many points are still obscure.

Intrathoracic Newgrowths and Bronchoscopy.

V. E. NEGUS (*The Journal of Laryngology and Otology*, July, 1933), in a paper dealing with the value of bronchoscopy in the diagnosis and treatment of intrathoracic newgrowths, gives the results of his experience of a number of bronchoscopic examinations of pulmonary affections, particularly neoplasms. Practically all the newgrowths encountered were malignant. Details of symptoms and physical signs are given, as also are examples of cases in which the aid of the bronchoscope has enabled difficulties of interpretation of physical signs and X ray appearances to be overcome. The author considers that in all cases of doubt bronchoscopic examination will be found most useful. A definite diagnosis should be made in every case, and decided improvement will often result from dilatation of the obstructed bronchi and the aspiration of the retained secretion. By bronchoscopic examination the presence or absence of occlusion of a bronchus is revealed, either by a circumspect or diffuse growth or by pressure from without; redness and swelling of the bronchial walls causing lack of aeration; and the presence of any pent-up secretions below the point of blockage. A piece of tissue can be removed if desired. There is no real contraindication to bronchoscopy.

The Effects of Drugs on Vestibular Reactions.

ELLISON L. ROSS AND AXEL OLSEN (*Archives of Otolaryngology*, December, 1933), to test the effects of drugs on vestibular reactions, gave to a series of animals with but one labyrinth, drugs representing the common pharmacological groups. Pentobarbital sodium represented the cerebral depressants; caffeine, cerebral stimulants; cocaine and epinephrine, stimulants of the sympathetic nervous system; and strychnine, stimulants of the cord. The change in the duration of the nystagmus after a standard rotation and quick stop was taken as the measure of the change in the irritability of the vestibular system. The cerebral depressants decreased the reaction. However, the estimated degree of general depression was as great as, or greater than, the decrease of the vestibular reaction. Caffeine did not increase the reaction to any significant degree. Stimulation of the sympathetic nervous system by cocaine and epinephrine had no significant effect. Strychnine, stimulating the spinal cord, decreased the vestibular reaction approximately 20%.

Fractures of the Larynx.

H. O. GARDNER (*Archives of Otolaryngology*, October, 1933) discusses fracture of the larynx and reports a case. He concludes that fractures of the larynx are not common because of the mobility and the protected position of the larynx. Diagnosis is difficult in many cases, and skiagrams are of little value. The mortality is high and probably in the neighbourhood of 30%. Palliative treatment may be warranted occasionally, but it is far safer to perform tracheotomy in all cases. Laryngofissure and laryngotomy may be necessary in some cases.

Severe Deafness in Adults.

GEORGE E. SHAMBAUGH *et alii* (*Archives of Otolaryngology*, October, 1933) have made a study of one hundred and sixty-five adults suffering from deafness severe enough to constitute a handicap. Thirty were afflicted with primary nerve deafness, caused by a congenital defect in two, by toxic neuritis from an acute infectious disease in five, by a noisy occupation in two, and by senile changes in seven. Syphilis was the probable cause in one case. In twelve patients with primary nerve deafness the cause was unknown, but in six of these the labyrinthine type of otosclerosis was suspected. Twenty patients suffered from a tubo-tympanic middle ear form of deafness. One hundred and fifteen patients had conditions diagnosed as otosclerosis with primary fixation of the stapes. The authors conclude that only 12% of severe adult deafness is due to middle ear disease beginning in childhood and is in any way preventible by examination of school children. Severe deafness in adults profound enough to constitute a handicap is due to otosclerosis with ankylosis of the stapes in the majority of instances (70%) and is neither preventible nor amenable to treatment in the present state of knowledge.

Primary Malignant Neoplasms of the Maxillary Sinus.

KARL MUSSER HOUSER (*Archives of Otolaryngology*, November, 1933), in a paper dealing with the diagnosis, treatment and end-results in 21 cases of primary malignant tumour of the maxillary sinus, states that the symptoms of early lesions are practically nil. The tumour must be fairly large before it gives rise to a sense of fullness in the antrum and must erode the bony wall of the sinus before tumefaction of the soft tissues of the cheek occurs. With growth, however, the initial symptom is pain. The author concludes that early diagnosis of antral neoplasms is necessary for the best therapeutic results. If an antral neoplasm is suspected, the sinus should be opened for inspection and biopsy. Surgical procedures should have as their major purpose exposure of the lesion, so that direct irradiation may be used. Surgical procedures are necessary for the maintenance of adequate drainage. Röntgen and radium therapy in adequate dosage offers the most hope of cure.

Involvement of the Œsophagus in Acute and Chronic Infection.

HARRIS P. MOSHER (*Archives of Otolaryngology*, November, 1933), in a paper dealing with involvement of the œsophagus in acute and chronic infection, gives instances of infection of the œsophagus itself from within. He also shows that infection of the œsophagus may occur in acute disease. In addition to the old finding that fibrosis of the œsophagus, especially of the terminal portion, is the result of infection from contiguous organs, it has been shown that fibrosis of isolated areas is fairly common, especially in such chronic infections as arteriosclerosis. In infections of the blood stream, the œsophagus is often involved to the extent of ulceration. In acute infections (pneumonia) the œsophagus may be infected. Chronic infection, as shown by an infiltration of lymphocytes under the epithelium and in the glands and about the gland ducts, is also common. Dilatation of the subepithelial blood vessels is almost constant in diseases such as cirrhosis of the liver which impede the venous circulation. The author has stated in previous papers that cirrhosis of the liver and infection of the gall-bladder are among the chief causes of infection of the œsophagus. As autopsy specimens of these conditions accumulate, this fact is becoming more and more evident. Hæmorrhage into the muscular layers extensive enough to disrupt them may occur when there is back pressure on the œsophageal vessels. The glands of the œsophagus are especially liable to infection, and are probably the chief route by which the œsophagus is infected from within. One form of solitary follicle is simply a collection of lymphocytes following a gland duct to the surface. The œsophagus, to recapitulate, can be infected from within and from without, and is often infected in both acute and chronic diseases. In the œsophagus, fibrosis follows infection, as it does in other organs of the body. As the œsophagus is so frequently infected, there is abundant cause for strictures or fibrosis to occur in any part. The concluding portion of the paper deals with the fluoroscopic examination of the œsophagus and the passage of the barium bag.

Special Report.

A REVIEW OF THE EVIDENCE RELATING TO LEAD AS AN ÆTIOLOGICAL AGENT IN CHRONIC NEPHRITIS IN QUEENSLAND.

THE following is a report by Keith D. Fairley, M.D., M.R.C.P., of the Walter and Eliza Hall Institute of Research in Pathology and Medicine, on the relationship of lead to chronic nephritis in Queensland. Reference is made to this report in the leading article in this issue.

In people under middle age, nephro-vascular diseases are much more common in Queensland than in New South Wales or Victoria. From this age onwards, the incidence of these disorders is roughly identical in all three States (Nye[1]). This is shown in Table I.

The present review of the evidence relating to chronic nephritis in Queensland was made to answer the following questions:

A. Is there sufficient evidence incriminating lead paint as a causative factor in chronic nephritis in Queensland?

B. If not, what further investigation is necessary in order to arrive at final conclusions in respect to the first question?

The first question involves answers to the following questions: (i) Does lead poisoning occur in Queensland children? (ii) Is it followed by chronic nephritis? (iii) Does it occur with sufficient frequency to be a factor in the extra mortality from chronic nephritis under the age of forty years? (iv) Is lead poisoning in Queensland children due to the ingestion of lead paint?

These questions will be considered in turn.

(i) DOES LEAD POISONING OCCUR IN QUEENSLAND CHILDREN?

There can be no doubt that lead poisoning does occur in children in Queensland, as was first pointed out by Lockhart Gibson[2] and Jefferis Turner,[3] the symptoms in many cases being comparable with those observed in industrial lead poisoning. This applies to (*a*) cases of symmetrical paralysis of gradual onset involving the extremities, (*b*) cases with colic and constipation, (*c*) certain cases of encephalopathy with convulsions. Lockhart Gibson[2] has described cases of "ocular neuritis" with gross papilloedema and paralysis of ocular muscles. There are also cases with atypical manifestations in which, failing complete investigation, the diagnosis remains uncertain.

(ii) IS LEAD POISONING IN QUEENSLAND CHILDREN FOLLOWED BY CHRONIC NEPHRITIS?

The only available figures relating to this question are those of McCann in the Federal Report[4] and of Nye.[1] In 100 cases of chronic nephritis in persons under forty years of age who were born in Queensland, McCann found 30 with a history of classical lead poisoning of the paralytic type and 16 with a history of indefinite plumbism. Nye, in a similar series of 186 cases (though apparently not all were born in Queensland), found 33 who had been treated for lead poisoning in childhood. In 1931 McCann followed up 28 patients who had been treated for lead poisoning with paralysis between 1915 and 1922 at the Hospital for Sick Children, Brisbane. By this time, 19 of these 28 patients had developed chronic nephritis. Of 34 patients seen by Nye some years after they had suffered from paralysis due to lead poisoning, 29 had developed chronic nephritis. Others of these two groups may later develop chronic nephritis. The incidence of chronic nephritis as a sequel of severe lead poisoning in childhood is therefore very high.

(iii) DOES LEAD POISONING OCCUR WITH SUFFICIENT FREQUENCY TO BE A FACTOR IN THE EXTRA MORTALITY FROM CHRONIC NEPHRITIS UNDER THE AGE OF FORTY YEARS?

In McCann's[4] series of 100 cases of chronic nephritis 30 patients had suffered from classical lead poisoning in childhood. Even if the diagnosis had been incorrect in some of the 16 others in whom the evidence of lead poisoning for which they were treated in childhood was not indisputable, this error might be counterbalanced by those who suffered from minor manifestations of lead poisoning in which the diagnosis was overlooked. This argument would also apply to Nye's series. It would appear, therefore, that lead poisoning occurs with sufficient frequency to be a factor in the extra mortality from chronic nephritis under the age of forty years in Queensland.

From 1917 to 1929 the average yearly mortality from chronic nephritis in people between the ages of ten and forty years in Queensland was 34·7 per 100,000 of the population between these ages (on the basis of the 1921 census figures). The corresponding mortality in New South Wales, Victoria and South Australia was 10·4, 10·7 and 9·6. Since the population in Queensland between the ages of ten and forty years was practically 400,000, these figures indicate that approximately 100 extra deaths from chronic nephritis occurred each year in the Queensland population between these ages. The average age at death from chronic nephritis following severe lead poisoning is roughly twenty years after the poisoning. Hence, if the majority of these extra 100 deaths per year in Queensland between 1917 and 1929 were due to lead poisoning, a yearly average of at least one hundred and probably some hundreds of cases of lead poisoning should have occurred in Queensland children between 1897 and 1909, when the population was much smaller than it now is.

The admissions to the Hospital for Sick Children, Brisbane, of patients with plumbism from 1898 to 1905 numbered 124 (15·5 per year), while from 1915 to 1930 they numbered 349 (22 per year), excluding readmissions. Of these 349 cases, 122 had typical major signs of lead poisoning. No allowance is made in the earlier figures for readmissions of the same patient or for errors in diagnosis. Unless minor degrees of plumbism, often unrecognized, have been responsible for many cases, it is clear that lead poisoning accounts only in part for this extra mortality from chronic nephritis.

Croll[5] showed that in Queensland there was in every decade a death rate from acute nephritis practically double that found in other Australian States. This may be due to an increased incidence of acute diffuse nephritis, an increased case mortality, or, as Croll suggests, the statistics may be unreliable owing to the inclusion of many cases of chronic nephritis. If there is an increased incidence of acute diffuse nephritis this may be a factor in the increased mortality from chronic nephritis. The type of nephritis under review apparently does not originate in an attack of acute diffuse nephritis, for a

TABLE I.

Approximate Comparative Figures of the Average Death Rate per 100,000 of Population in Different Decades, taking One State as Unity in each Decade. Years 1916-1931 inclusive.

Years.	0-10.	10-20.	20-30.	30-40.	40-50.	50-60.	60-70.	70 and Over.
Queensland	2·5	3	3	2	1	1·00	1·0	1·0
New South Wales ..	1·0	1	1	1	1	1·10	1·2	1·3
Victoria	2·5	1	1	1	1	1·15	1·2	1·3

history of acute nephritis or of hæmaturia in childhood was found in only five of 286 cases of chronic nephritis reviewed by Nye[3] and McCann.[4]

On clinical grounds, which are admittedly unsatisfactory, McCann considered that 62% of his cases were of the type of the arteriosclerotic kidney (such as might result from lead poisoning), 13% were of the type of chronic glomerulonephritis (such as would originate from an acute diffuse glomerulonephritis) and 25% could not be classified on the evidence available. Unfortunately, published histological evidence is meagre. The results of a series of complete *post mortem* examinations with the histological findings would provide valuable data which should be correlated with the results of the analysis of portions of the skeleton for lead, though the finding of lead in these circumstances would merely prove its absorption. As a control, a series of patients of the same age dying from other causes should be similarly investigated.

Any damage to the kidney may render this organ more liable to further injury by noxious agents (such as lead) which are exercised by the kidneys. Scarlet fever, syphilis and hereditary influences have been instanced in the Federal Report[40] as possible factors producing the initial kidney defects.

The available evidence fails to show that nephritis from scarlet fever is more common or more severe in Queensland than in the other Australian States, and as mentioned previously, a history of preceding acute nephritis is very rarely obtained in cases of chronic nephritis in Queensland.

There is no evidence that other infections, such as diphtheria, measles, antral and tonsillar infections, bronchiectasis, malaria and filariasis play any important part in the production of the extra mortality.

A history of congenital syphilis was found in only one of 286 patients with chronic nephritis in the combined series of McCann[4] and Nye.[3] In Nye's series "the number of our nephritic patients who gave a positive reaction to the Wassermann test was no greater than would be found in the ordinary population". In 6 of 35 cases in McCann's series a positive Wassermann reaction was obtained, as it was in 5 of 47 patients with chronic nephritis at the Brisbane General Hospital. This incidence of syphilis (approximately 13%) is comparable with that found by Hamilton Fairley and Fowler[38] in Victoria. The mothers and younger members of four Queensland families with a high mortality from chronic nephritis were found to give positive reactions to the Wassermann test.[4] This suggests, but does not prove, that the older members who had chronic nephritis also suffered from congenital syphilis. At the most, syphilis plays only a very minor part in the increased incidence of chronic nephritis in Queensland.

In any series of cases of chronic nephritis in young adults there will be a certain proportion with a history of this disease in the parents or grandparents, since chronic nephritis is not an uncommon cause of death in later life. That 10 (5%) of 200 parents (McCann[4]) should suffer from this complaint is a proportion in excess of what might be expected. That only two of 400 grandparents were known to have died from this disease suggests that details of the cause of death were often unknown. McCann[4] found that 24 of his 100 patients had one or more brothers or sisters with chronic nephritis. This may be related to hereditary influences or to common environmental factors, or to both of these conditions. The influence of heredity is by no means clear and the occurrence of chronic nephritis in several members of families may fairly be attributed to a common noxious agent.

(iv) Is Lead Poisoning in Queensland Related to the Ingestion of Lead Paint from Houses?

The answer to this question may be considered in three parts: (a) What is the source of the lead? (b) What factors determine the lead intake? (c) What factors determine the occurrence of acute or chronic plumbism in individual cases?

iv (a) What is the Source of the Lead?

Several possible sources of lead have received consideration in the past and most of these have been discarded. The original suspicion that the source of the poisoning might be the tinfoil wrappings of sweets and cigarettes was soon disproved, nor could it be traced to ginger beer, cooking utensils, jam tins, or painted toys. In Japan numerous cases of lead encephalopathy in breast-fed infants have occurred for over two centuries, and in 1923 it was shown that the source of the poisoning was white lead used in toilet powders applied by nursing mothers to their breasts or to the skin of the infants. In the United States of America a few cases of lead poisoning in infants and children have been reported, usually in those with a perverted appetite (pica) who ate the lead paint off their cots, toys or other articles. Occasional cases have resulted from the use of ointments containing lead in the treatment of eczema of the nipples of nursing mothers. A recent case at the Children's Hospital, Melbourne, was due to the eating of a certain amount of "Bates' salve" (an ointment containing lead) by a child suffering from pica. There is no evidence that lead poisoning in Queensland children is related to any perversion of appetite.

The three main sources that have been suggested for the ingestion of lead in Queensland children are: (i) drinking water, (ii) vegetables that have been sprayed with lead arsenate, (iii) lead paint used on external surfaces of houses, especially veranda railings.

(i) *Drinking Water.*—Children apparently cured in hospital frequently relapsed within a short time of their return to the former living conditions at home. Enoggera water, the only water with any plumbi-solvent properties supplied in Brisbane, was supplied to the Children's Hospital, Brisbane, till 1906 (Bush[37]), yet even before the water supply was changed patients in this hospital recovered rapidly from their poisoning. Lockhart Gibson[39] draws attention to this fact. It seems unlikely, therefore, that they were ingesting any appreciable amounts of lead with this water. Also children confined to bed in their own home for these symptoms would improve and were prone to relapse again when they were once more up and about. These facts suggest that the source of the lead is at home and is available to the children only when they are up and about. It appears that drinking water and vegetables are not the source of lead, since they would be available when the children were confined to bed. Recent cases of lead poisoning are not restricted to the diminishing area supplied by Enoggera water, which since 1921 has rarely shown plumbi-solvent properties (Chamberlain[36]).

A study of the water supplies of other towns in Queensland by Nye[3] revealed no evidence that plumbi-solvent properties in the water supply were associated with plumbism or chronic nephritis.

The water from galvanized iron tanks always contains zinc, which will react with lead carbonate, precipitating insoluble metallic lead. In country areas, where the main water supply is from such tanks, lead poisoning is almost unknown.

(ii) *Vegetables Sprayed with Lead Arsenate.*—Vegetables sprayed with lead arsenate may be a source of occasional lead poisoning. Certainly, from the figures given in the Federal Report[40] from the Annual Report of the Commissioner of Public Health to June 30, 1931, this appears possible. "Of 31 samples of cabbage, 29 showed from 3 to 20 grains of lead arsenate per cabbage." Though the outer leaves are discarded before cooking and the cabbage is usually soaked in water before being transferred to fresh boiling water, the preparation of such vegetables for the table probably does not remove all the poison. This could easily be proved by an analysis of a number of cabbages before and after their cooking. The cases in which poisoning arose from lead arsenate in this manner would presumably show the presence of arsenic in the urine and fæces and in the hair. Such an analysis would be advisable in a series of cases of lead poisoning in children, and if arsenic were present it would suggest the advisability of further legislation regulating the use of lead arsenate as a spray for

vegetables and fruits. A new by-law relating to poison sprays under the *Health Act*, 1911-1926, in Western Australia prohibits the application of arsenate of lead either as a spray or a powder to cabbage, cauliflower or any other plant of the Brassica family within four weeks of cutting.

Since lead arsenate has only been in use for about twenty years, and lead poisoning has been frequent in Queensland children for over forty years, it is obvious that although this may be a factor in some cases it is not the main source of lead poisoning.

(iii) *Lead Paint.*—A large proportion (stated to be 86%) of houses in Queensland are wooden and many of these are painted with lead-containing paints. Experiments by various observers have shown that this paint when desiccated provided a widely accessible supply of soluble lead carbonate, adequate, if ingested, for the production of lead poisoning in children.

A series of experiments (Federal Report[a]) showed that the loose surface layer of weathered house paint removed by a dry rub always contained large quantities of lead, the fraction soluble in 1% acetic acid (oxide and carbonates) containing from 50% to 60% of lead. The amount of lead adhering to an adult hand deliberately thickly coated by contact with a wooden surface covered with very dry powdery lead paint varied from 0·0145 to 0·0431 gramme (one-quarter to two-thirds of a grain approximately). The area of the hand covered was approximately 68 square centimetres (11 square inches). Jefferis Turner[aa] states that the palms of the hands are covered with white powder in two minutes by rubbing them against veranda railings coated with powdery paint. It is obvious that the young child may get large amounts of such paint on his hands in the course of weeks or months. Lead is therefore accessible to young children in poisonous quantities from this source. In 11 of 58 cases of definite plumbism reviewed in the Federal Report[a] the actual ingestion of considerable quantities of lead paint was casually related to the occurrence of lead poisoning in childhood, and in the remaining cases of this series lead paint was an accessible source for the poisoning. There is obvious justification for the claim that many cases of lead poisoning in Queensland children are derived from lead paint.

(iv) (b) What Factors Determine the Lead Intake?

Factors determining the lead intake are:

1. The availability of lead for ingestion. Lead is available for young children when they are confined for many hours each day over a period of weeks or months to verandas, the railings of which are covered with weathered lead paint.

2. Habits resulting in the ingestion of lead. From the hands lead may be ingested if the child is in the habit of biting his nails or sucking his fingers or thumbs. Failure to wash the hands before eating may also be a factor of some importance. Habits of eating the powdered paint or of licking raindrops from veranda railings may be responsible in some instances for the ingestion of lead.

The hypothesis advanced with regard to the part played by weathered lead paint as the source of poisoning may be summarized as follows: Most of the wooden houses in Queensland are raised on piles and have large verandas which necessarily have railings and a gate which are painted. Paint weathers rapidly in the usual Queensland climate, with its high average temperature, high average wind velocity and long hours of sunshine. Particularly in the hot wet season, and to a less extent throughout the remainder of the year, infants and young children are confined to these verandas for long periods day after day. This applies from the crawling age to the school age. When the paint on the railings is dry and powdery, the hands of these children each day may become covered with powdered paint. Those with the habits mentioned above will ingest lead in at least small amounts almost daily over a prolonged period. Many of them in time will show evidence of lead poisoning. In certain cases the sources of lead may be other painted external surfaces, such as fences.

Any theory with regard to the source of lead poisoning in Queensland children must account for the following conditions:

1. The age incidence of lead poisoning in Queensland.
2. The frequency of these cases in Queensland and their extreme rarity elsewhere.
3. The apparent limitation of these cases to certain towns in Queensland.
4. The decreased incidence of these cases in recent years.
5. The relative rarity of cases despite the fact that from many of the houses weathered lead paint can be brushed off easily.
6. Why in some instances only one member of a family is affected and in others several or all members may suffer.

1. *The Age Incidence of Lead Poisoning in Queensland.*

McCann[o] found that the illness commenced at or under four years of age in 66% of the fifty cases with paralysis examined by him, and in practically every instance where a history was obtainable from birth, the symptoms attributable to lead poisoning began not later than the sixth or seventh year. He concluded that when industrial causes were excluded, lead poisoning was confined to children under nine years old. The records of the Children's Hospital, Brisbane, show that the age of onset is usually between eighteen months and three years. Apparently no case has been recorded before the crawling age. Cases in children under school age are readily explained by the veranda theory. Some cases from five to eight years of age may be due to persistence of nail-biting and similar habits in those children who have continued access to weathered lead paint. Others may be due to the mobilization of lead stored in the skeleton at an earlier age. Anything that tends to mobilize calcium from the bones may have this effect. Cases of "ocular neuritis" are found in this same age period (two to eight years), though they are apparently more common from four to eight years old. Lead encephalopathy is frequently due to the ingestion of relatively large amounts of lead over a short period; a single large dose of a lead salt may cause this. It appears likely that at least some of these cases in the children of school age may be due to the rapid mobilization of lead previously stored in the bones. The age incidence of lead poisoning in Queensland children is explicable in terms of the "veranda" theory.

2. *The Frequency of these Cases in Queensland and their Extreme Rarity Elsewhere.*

Paint weathers in any climate, and powdered paint must be accessible in some degree to children in many countries. Nail-biting and finger-sucking are common in children everywhere. The occurrence of poisoning depends mainly on the quantity of lead ingested, and this is related to two chief factors: the domestic architecture and the habit of confining young children for long hours day after day on verandas. The wooden houses in Queensland, with extensive verandas railed off because the houses are usually raised on piles, are uncommon elsewhere. The climatic conditions are such that young children are confined for hours, day after day, over many months of the year, to these verandas where powdered paint is often accessible. These two factors seem sufficient to explain the increased lead hazard and the resulting common occurrence of lead poisoning in Queensland children.

Other conditions may also be of importance. Workers in lead suffer from plumbism more frequently during hot months (Gwyn[ap]), as do the children in Queensland. The tropical climate may increase the liability of children to lead poisoning. Sundstroem[aq] showed that inhabitants of the tropics tended to develop an alkalosis. The administration of alkalis (sodium bicarbonate) is used therapeutically to mobilize lead stored in the skeleton. The mobilization of lead secondary to an alkalosis may be a factor in the increased incidence of lead poisoning in Queensland children during the summer months. Cilento[ar] stated that malnutrition of various types was not uncommon in Queensland. If calcium deficiency were common in these

children, lead would not be stored readily or would tend to be mobilized if it had been stored previously in the bones.

3. *The Apparent Limitation of these Cases to Certain Towns in Queensland.*

In the Federal Report[4] it is stated that "the outstanding fact against lead poisoning as a sequel of the desiccation of paint on houses has always been that the condition appears to be restricted to Brisbane and to two or three large cities in Queensland, while elsewhere, where conditions and climate appear to be similar, evidence of any involvement is lacking".

In an historical account of lead poisoning in Queensland in 1922[13] it is stated that by 1897 cases had occurred mostly in Brisbane children, but also at Charters Towers, Gympie and Southport. In 1899 cases were recorded from Maryborough, Ipswich, Toowoomba, and two cases were observed from New South Wales. A series of sixteen cases from Townsville were reviewed by Breinl and Young[14] in 1914. Cilento[15] states that a typical case of lead poisoning was seen in 1931 at Rockhampton in a child aged four years, and occasional cases have been seen recently in Townsville. An analysis by Nye[1] of cases of typical major manifestations of lead poisoning admitted to the Children's Hospital, Brisbane, between 1914 and 1931 shows that they came from very many different parts of Brisbane and its suburbs, while outside the metropolitan area the following townships were recorded: Beenleigh, Biggenden, Bundaberg, Caboolture, Deagon, Graceville, Manly, Maryborough, Rocklea, Roma, Sandgate, Sherwood, Southport, Townsville, Toogoolawah, Torwood, and Warwick. Cases undoubtedly occur elsewhere in Queensland, but are often not reported because of insufficient investigation. Statistics would be obtained by making lead poisoning in children a notifiable disease, though their reliability would be subject to criticism owing to the difficulty in diagnosis of the minor manifestations of lead poisoning.

Nye[1] states that the absence of lead poisoning in rural districts is due to the fact that the children are not confined for long hours to verandas as they are in large towns, where the dangers of traffic necessitate that they shall be prevented from wandering.

4. *The Decreased Incidence of these Cases in Recent Years.*

A variety of factors is responsible for the decreased incidence of severe plumbism in Queensland children in recent years. These have been fully discussed by Nye.[1] Though no case with paralysis from lead poisoning was admitted to the Children's Hospital during the period covered by investigations summarized in the Federal Report (1930-1931), typical cases of such paralysis have been occurring in the past few months, seven severe cases being under observation in Brisbane within three months in the latter half of 1933.

Some of the factors responsible for this decrease may be summarized as follows:

A. Earlier diagnosis by the medical profession.

B. The decrease in the amount of desiccated paint containing lead available for ingestion due to: (*a*) the frequent use of non-poisonous paints, (*b*) legislation against the use of lead paint on veranda railings, (*c*) the education of the public regarding the dangers of poisoning from paints containing lead, (*d*) the more frequent painting of houses during the period of post-war prosperity.

C. Changes in the management of children tending to lessen the exposure to desiccated paint: (*a*) improved hygiene in the home and at school, (*b*) the work of the Creche and Kindergarten Society.

Some of these factors will be discussed briefly.

A. Earlier Diagnosis by the Medical Profession.—Badham[12] has been impressed with the rarity of cases of paralysis due to lead poisoning of recent years in workers in industries where there is exposure to lead. This is partly due to the introduction of methods reducing the lead hazard, but largely to the increased medical care received by these workers. Cases of lead poisoning are diagnosed in the relatively early stages, when there are only minor blood changes (mild anæmia and a moderate number of red cells showing basophilic stippling) and perhaps lead colic. Removal from the source of lead then is usually sufficient to allow the patient to recover without the development of any paralysis. The great interest taken by the medical profession in Brisbane during the last decade in the question of plumbism undoubtedly has resulted in earlier diagnosis in many cases and must have led to an appreciable diminution in the number of patients who otherwise would have developed major manifestations of lead poisoning.

B. The Decrease in the Amount of Desiccated Paint Containing Lead Available for Ingestion.—In the Federal Report[4] figures from the analysis of twenty-seven brands of paint ordinarily on the market are given. Only two of ten samples of so-called lead-containing paints contained more than a negligible amount of lead, while three of seventeen paints labelled non-poisonous contained over 50% of lead. That only five out of twenty-seven paints ordinarily on the Queensland market should contain lead in more than negligible amounts is an indication of the widespread use of non-poisonous paints. Presumably this is largely due to education of the public regarding the potential dangers of lead paints and to legislation restricting the use of such paints. In the period of post-war prosperity, houses would probably be painted more frequently, and so there would be less desiccated paint available to children. The present economic depression would have the reverse effect, and this may be a factor in the recent increase in the number of cases seen in Brisbane.

5. *The Relative Rarity of Cases Despite the Fact that from Many of the Houses Weathered Lead Paint can be Brushed Off Easily.*

The infrequency of lead poisoning in children apparently exposed to this lead hazard is the most difficult fact to reconcile with the veranda theory. According to this theory for lead poisoning to occur in young children from the ingestion of weathered paint containing large amounts of lead, the following conditions must be present:

(i) The veranda railings must be coated with weathered paint containing lead in large quantities.

(ii) This must be present over a period of weeks or months when children between the ages of two and eight years, and especially between two and four years of age, are in the house.

(iii) These children must be confined for some hours, day after day, over a period of weeks or months to the veranda.

(iv) The children must have developed some habit (such as nail-biting, thumb sucking, eating the powdered paint, or licking rain drops from the veranda railings) which will result in the ingestion of powdered paint.

The majority of children who develop plumbism in Queensland bite their nails or suck their fingers. In 1,000 consecutive children attending State schools, 13·7% were found[1] to have the habit of biting their nails. A conservative estimate of the incidence of the above habits which lead to the ingestion of lead paint by children would probably give a figure of one child in six so affected. Since the incidence of manifest plumbism in Queensland children is far removed from such a figure it would appear that other factors must be of great importance. Weathered paint is apparently commonly available in Queensland over these critical years of the child's life from two to four years of age. Of the factors mentioned above, the most variable one which might explain the relatively low incidence of plumbism would appear to be the hours of confinement of the young children to verandas. The requisite data concerning this factor would be very difficult to obtain with any degree of accuracy.

If this variable does not explain the comparatively low incidence of manifest plumbism in Queensland children,

there must be some secondary factor or factors increasing the susceptibility of individual children to lead poisoning. These possible factors have been discussed previously—conditions such as alkalosis, calcium deficiency, lowering of resistance to lead poisoning by previous disease, and so forth. There is some evidence that an increased susceptibility to the effects of lead may be inherited.

The available evidence shows that plumbism is of vastly greater frequency in nail-biters and thumb-suckers than in children without these habits, which apparently precede the ingestion of lead and are not effects of lead poisoning. A survey of a large series of young children with these habits who have been exposed for hours day after day over long periods on verandas containing weathered lead paint would provide evidence of importance, for they would presumably all show the presence of lead in the fæces to a much greater degree than a control series who were confined to verandas painted with non-poisonous compounds. This, of course, would merely show an increased ingestion of lead, but a complete survey might reveal a much greater proportion with evidence of lead absorption (blue line on gums or lead in urine or in bones or teeth) and of minor and major manifestations of lead poisoning than would be found in the control series. Similar comparisons might be made in two groups of children who have not developed those habits considered to lead to the ingestion of lead.

Aub and his co-workers[36] have made a few estimations of the amount of lead to be found in extracted teeth of patients with lead poisoning. Their results show that "a concentration of lead similar to that found in the rest of the bony tissue is present in teeth". They suggest that such an analysis of extracted teeth is a possible method of proving previous lead absorption, since lead stored in the skeleton remains there long after exposure to lead has ceased. The analysis of extracted milk teeth of children in these various groups might prove to be a satisfactory method of showing how widespread is the exposure of Queensland children to the lead hazard. The presence of lead in the teeth merely proves that lead absorption has occurred at some earlier date. Lead should be present in the teeth of young children who have previously absorbed lead even when at the time of examination no lead is being excreted in the fæces or the urine. Similar evidence may be obtained by radiological examination of the ends of the long bones. This frequently shows that lead has been deposited in this situation.

Badham[35] considers that, in workers in New South Wales, the detection of basophilic stippling of the red blood corpuscles in the absence of signs of a primary anæmia or of a good cause for a secondary anæmia (or even in their presence, if the number of affected cells is large) is presumptive evidence of an intake of lead, requiring investigation of the industrial conditions and the lead excretion of the particular patient. He states that punctate basophilia is not found in the blood of normal persons in Sydney, and he regards this abnormality resulting from the action of lead as the earliest indication of lead poisoning, whether any disability be present or not. He work was done on industrial workers, but the conclusions probably apply also to children. Were a complete investigation of Queensland children on the lines mentioned above considered necessary, this point would receive attention. At the same time, a control series of children of similar ages in another Australian State should have their blood investigated by the same technique for basophilic stippling of the red cells. Since in Queensland opportunities for lead ingestion are so widespread, a considerably larger proportion of apparently normal Queensland children would probably show this reaction as compared with the control group. Badham[35] believes that the punctate basophilia reported by various observers as occurring in so-called normal persons comes from the ingestion of lead. This punctate basophilia resulting from the action of lead disappears more or less rapidly after removal of the patient from the lead hazard.

The variables mentioned above may be sufficient to explain the comparatively low incidence of manifest lead poisoning in children. Minor manifestations of lead poisoning may also be more common in Queensland children than is usually imagined. A fair proportion of the young children in Queensland must be exposed to these conditions, and if they are, apparently at least one in every six so exposed is ingesting variable amounts of lead, though relatively few develop major manifestations of plumbism. Individual susceptibility is presumably important. It is sufficiently common and the effects of lead poisoning in childhood are so serious that every effort should be made to prevent these susceptibles from ingesting lead. Since the factors underlying susceptibility are unknown, the removal of the source of lead appears to be the only method of achieving this object.

6. *Why in Some Instances Only One Member of a Family is Affected and in Others Several or all Members of a Family May Suffer.*

The factors discussed above with regard to the availability of weathered lead paint, the varying duration of access to such paint, the variability regarding habits leading to the ingestion of lead paint and questions of individual and familial susceptibility to lead poisoning, appear to be sufficient to explain this varying incidence in different families.

(iv) (c) What Factors Determine the Occurrence of Acute or Chronic Plumbism in Individual Cases?

The extreme variation in individual susceptibility to lead poisoning is well recognized. Children are more susceptible than adults, and females are more susceptible than males. Acute plumbism is seen with relatively large doses of lead. This is not infrequent in women who have used pills made from diachylon plaster as an abortifacient. The sudden flooding of the organism with large amounts of lead poisoning stored in the bones of a patient who has been exposed for long periods to a lead hazard may also result in symptoms of acute plumbism.

Chronic plumbism may result from the exposure to small doses of lead over a prolonged period. Some of these patients may develop definite colic or paralysis typical of lead poisoning; many will show only minor manifestations and the diagnosis will frequently be overlooked.

The factors underlying variations in individual susceptibility to lead poisoning are unknown. Intercurrent infections, states of acidosis or alkalosis, dietetic defects and abnormalities of calcium metabolism leading to a mobilization of calcium from the bones, are some of the many conditions which increase the liability to lead poisoning. These considerations apply whatever the route by which the lead is introduced into the organism. Where lead is ingested, the additional factor of individual variation in the functional capacity of the liver must also be of importance.

Legislation in Reference to Lead Poisoning.

Hoffmann[37] in a review of legislation regarding lead poisoning states that 18 countries (Austria, Belgium, Bulgaria, Chile, Cuba, Czecho-Slovakia, Estonia, Finland, France, Greece, Jugo-Slavia, Latvia, Luxembourg, Norway, Poland, Roumania, Spain and Sweden) have adopted the International Convention regarding the use of white lead in painting. Each member of the Convention agrees to prohibit, with certain exceptions, the use of white lead and lead sulphate and of all products containing these pigments in the internal painting of buildings. The use of white pigments containing not more than 2% of lead expressed in terms of metallic lead is permitted. The prohibition does not apply to artistic painting or fine lining. The employment of males under eighteen years of age and of females is prohibited in any industrial painting involving the use of these lead compounds. All cases of proved or suspected lead poisoning are reported and subsequently verified by a medical man appointed by a competent authority.

Britain has not adopted the convention, but has passed adequate regulations. In Switzerland the subject is being

seriously considered. Hoffmann's address contains a translation of the report of Director Tzaut of the Swiss Accident Assurance Society, Lucerne (a government institution) to the Federal Commission on lead poisoning, May, 1929. Tzaut states that his institution has reports from leading industries to the effect that for all practical purposes, lead-free paint can be used as a satisfactory substitute for lead-containing paint. The managers of a large paint industry in Paris reported to this institution that for many years zinc paint had been used by them exclusively for both interior and exterior painting and they were fully convinced of the practical usefulness of lead-free paint for such purposes. Tzaut's institution "plans to make a number of technical studies of the durability of lead-free paint". Further information on this subject has presumably been collected by his institution in the last few years.

In Germany, all paints containing less than two per cent. of lead are considered innocuous. Lead-containing paints are prohibited for interior painting except for railways and large buildings for outdoor purposes. Rubbing off of paints by hand and dry rubbing are prohibited, as is the employment of women or males under eighteen years old. Eating, drinking and smoking during painting operations are prohibited. Separate working clothes are required, definite hygienic precautions are enforced, and employees must be medically examined every six months. The use of lead paints in Germany during the last 20 years has steadily declined.

The prohibition of the use of lead paint or the introduction of these stringent regulations controlling its use in these countries has been done to protect the adult painter from lead poisoning. The risk to the adult painter is apparently much less than the hazard to the child of prolonged contact with weathered lead paint.

Hoffmann's report contains references to eight deaths from lead poisoning in 1930 in children under four years of age in the United States of America. Seven were due to the ingestion of lead paint from furniture or other painted wooden surfaces. In one the origin of the lead was unknown.

Summary and Conclusions.

1. Chronic nephritis in people under 40 years of age is approximately three times as common in Queensland as in the other Australian States.

2. Typical lead poisoning occurs more commonly in Queensland children than in children elsewhere, except perhaps in Japan, where infants were poisoned from the use of toilet powders containing white lead. Typical severe lead poisoning is still being observed in Queensland children, and the notification of all cases of plumbism and suspected plumbism in children under the age of ten years would give information of some value. If all such cases could be investigated by a competent authority, the reliability of these statistics would be greatly enhanced.

3. A very large proportion of young children who suffer from lead poisoning sufficiently severe to cause paralysis die from chronic nephritis before middle age.

4. The increased mortality before middle age from chronic nephritis in Queensland is in part due to lead poisoning in childhood.

5. The arteriolosclerotic type of chronic nephritis is apparently responsible for the increased mortality in young people from chronic nephritis in Queensland. This type of nephritis arises independently of any preceding acute nephritis. The publication of the results of a series of complete *post mortem* examinations with the histological findings in cases of chronic nephritis in young people in Queensland would provide data of considerable value. An analysis of the skeleton for stored lead would give suggestive evidence of the relation of these cases to previous lead absorption. A control series of cases of about the same age would also have to be investigated.

6. Hereditary influences, scarlet fever, congenital syphilis and other infections may act in certain cases as factors predisposing to lead poisoning and chronic nephritis, but the available evidence does not indicate that they play any important part in the ætiology of chronic nephritis in young people in Queensland, except in so far as hereditary influences affect the individual susceptibility to lead poisoning.

7. Most cases of lead poisoning in Queensland children are apparently derived from weathered lead paint on external surfaces, chiefly veranda railings. Some may arise from the ingestion of fruit or vegetables that have been sprayed with lead arsenate.

8. The chief objection to the "veranda" theory of lead poisoning in Queensland children is the relatively low incidence of plumbism when large numbers of children are apparently exposed to this hazard. However, so many variable factors enter into this problem that it is impossible to determine accurately the individual exposure to lead. Furthermore the incidence of minor manifestations of lead poisoning may be much greater than is usually imagined. A comparison of the incidence of punctate basophilia in children in Queensland and in other Australian States would give valuable evidence regarding this. Scientific proof of the theory could only be obtained by comparing two groups of children from about two to six years of age who have the habits (nail-biting, thumb-sucking, *et cetera*) previously described as responsible for the ingestion of available lead from weathered paint. One group would be selected from houses with verandas coated with weathered lead paint, the other group would live in houses where the verandas were painted with non-poisonous pigments. At intervals over a considerable period, these groups would be subjected to an analysis of the fæces and urine and of any extracted teeth for lead, radiological examination of the ends of the long bones for deposits of lead, examination of the blood for basophilic stippling of the red corpuscles, and a thorough clinical investigation. The groups of children studied would require to be sufficiently large to exclude individual variability and variations in the other conditions to which the subjects were exposed. A similar comparison of two groups of children who have not developed these habits (nail-biting, *et cetera*) would be made at the same time. Such a complete investigation would not be justifiable, for in the light of the evidence already available, removal of the lead hazard would be indicated on humanitarian grounds. The available evidence points to weathered lead paint as the probable source of the majority of cases of lead poisoning in Queensland children. There is ample evidence that weathered lead paint on verandas in the homes of the industrial population provides a widely distributed supply of soluble lead carbonate readily accessible to young children.

The first question, "Is there sufficient evidence incriminating lead paint as a causative factor in chronic nephritis in Queensland?", is answered in the affirmative. The use of lead paint on veranda railings in Queensland exposes the young children to a definite lead hazard. Some develop severe lead poisoning and a large proportion of these die from chronic nephritis before middle age.

Legislation against lead paint should be directed against the use of this paint on any articles available to children—for instance, furniture, toys, pencils and so forth, as well as on external surfaces such as verandas. Partial prohibition is usually ineffective and gives rise to a false sense of security. To be effective, prohibition should be complete and unconditional to remove entirely the lead hazard. Stringent regulations should also govern the use of lead arsenate on fruit and vegetables, and should cases of poisoning from this source prove these restrictions ineffective, the prohibition of this poison should also be considered. In poisoning from this source, evidence of ingestion of arsenic as well as lead should be sought by the biochemist.

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(6) N. H. Fairley and R. Fowler: THE MEDICAL JOURNAL OF AUSTRALIA, August 20, 1921, page 150.

(7) W. E. Bush (Chief Engineer, Water Supply and Sewerage Department, Brisbane City Council): Personal communication.

(8) J. L. Gibson: Quoted by Nye.(9)

(9) W. J. Chamberlain: Quoted by Nye.(9)

(10) A. J. Turner: THE MEDICAL JOURNAL OF AUSTRALIA, April 1, 1922, page 366.

(11) N. B. Gwyn: Osler and McCrae, "Modern Medicine", Volume II, 1925, page 647.

(12) E. S. Sundstroem: University of California Publications, "Physiology", Volume VI, 1926, page 92.

(13) The Council of the Queensland Branch of the British Medical Association, THE MEDICAL JOURNAL OF AUSTRALIA, February 11, 1922, page 148.

(14) A. Brieni and W. J. Young: *Annals of Tropical Medicine and Parasitology*, Volume VIII, December 15, 1914, page 575.

(15) C. Badham: THE MEDICAL JOURNAL OF AUSTRALIA, December 16, 1933, page 816.

(16) J. C. Aub, L. T. Fairhall, A. S. Minot and P. Reznikoff: *Medicine*, Volume IV, February-May, 1925, page 1.

(17) F. L. Hoffmann: "Lead-Poisoning Legislation and Statistics", 1933.

Addendum.

Other articles of importance in the Australian literature in relation to this subject are the following:

C. Badham and H. B. Taylor: "Report of the Director-General of Public Health of New South Wales", 1925.
C. L. Chapman: "Report of the Royal Commission of Plumbism, South Australia", 1925.
S. F. McDonald: THE MEDICAL JOURNAL OF AUSTRALIA, May 9, 1931, page 572.
R. Y. Mathew: THE MEDICAL JOURNAL OF AUSTRALIA, February 16, 1929, page 208.
W. T. Nelson, L. Lockwood and K. Mackay: THE MEDICAL JOURNAL OF AUSTRALIA, September 10, 1932, page 317.
S. A. Smith: THE MEDICAL JOURNAL OF AUSTRALIA, March 11, 1922, page 284; September 26, 1925, page 391.
R. W. Tannahill: THE MEDICAL JOURNAL OF AUSTRALIA, February 16, 1929, page 194.

British Medical Association News.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE TASMANIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Tasmanian Museum, Hobart, on March 24, 1934, DR. W. K. MCINTYRE, the President, in the chair.

AN AUSTRALIAN SOCIETY OF ANÆSTHETISTS.

The Honorary Secretary read a letter from Dr. Geoffrey Kaye concerning the formation of an Australian Society of Anæsthetists. It was resolved that the letter should be sent to the Executive Committee of Congress to be dealt with.

ANNUAL REPORT OF COUNCIL.

The Honorary Secretary read the annual report of the Council. The report, which was adopted, is as follows.

The Branch Council has much pleasure in presenting the annual report for the year ending December 31, 1933, as follows:

The membership of the Branch, which was seventy at the end of last year, was eighty-four at the beginning of this year. Sixteen new members have been elected, seven have been transferred into the Branch, four have resumed membership by payment of overdue subscriptions within the period of grace. One member has resigned, four left the State, and eight members lapsed on January 1, 1934, through non-payment of their subscription for the year 1933; all but one of these members have since paid their 1933 subscription.

Meetings.

Since the last annual meeting there have been held ten ordinary and two special meetings of the Branch, the average attendance being 17·2, against 14·9 for the previous year.

Papers have been read by Dr. F. W. Fay, Dr. A. W. Shugg, Dr. R. Whishaw, Dr. Freeman, Dr. C. N. Atkins, and Dr. D. W. Parker.

Cases have been demonstrated or described by Dr. J. Stoddart Barr, Dr. E. Brettingham-Moore, Dr. T. H. Goddard, Dr. R. Whishaw, Dr. T. Butler, Dr. A. W. Shugg, Dr. Gibson, Dr. F. W. Fay, Dr. F. Short, Dr. T. Giblin, Dr. H. W. Sweetnam, Dr. J. H. B. Walch.

Eighteen meetings of the Branch Council have been held, the attendance being as follows:

Dr. W. E. L. Crowther	13	Dr. A. W. Shugg	12
Dr. S. Gibson	15	Dr. F. W. Fay	13
Dr. J. S. Reid	14	Dr. T. H. Goddard	15
Dr. J. H. B. Walch	18	Dr. C. N. Atkins	18
Dr. G. Sprott	5		

The northern members, Dr. W. K. McIntyre and Dr. G. E. Clemons, have not been able to attend.

Australasian Medical Congress (British Medical Association).

The outstanding activity of the Branch during the year has, of course, been the organization of the Fourth Session of the Australasian Medical Congress, held at Hobart in January of this year. In this connexion the Branch owes a great debt of gratitude to the many members who took part in the organization, and especially to the President of Congress, Dr. D. H. E. Lines, and the Joint Honorary Secretaries, Dr. H. N. Butler and Dr. W. L. Crowther, to whose arduous work the success of the session was due.

Anti-Cancer Campaign.

In accordance with instructions from the last annual meeting, the Branch Council considered the inauguration of anti-cancer activities in Hobart, but decided it would be unwise to initiate a campaign while members were occupied with the preparation for Congress. However, arrangements were made for the formation of preliminary articles for the public Press.

Medical and Hospital Insurance.

The Branch Council gave much time to detailed consideration of a scheme for medical and hospital insurance, proposed by the Colonial Mutual Assurance Society, and held a special meeting to discuss the matter with representatives of the Society.

Other Matters.

Other matters which have occupied the attention of the Council are: the conditions under which honorary medical service should be rendered to sporting clubs; the advisability of boys taking part in rowing races; the proposed legislation connected with the control of drug traffic.

Dr. Gregory Sprott.

Since last September the Branch lost the services of Dr. Gregory Sprott as a representative on the Federal Council and an *ex officio* member of the Branch Council. Dr. Sprott's resignation from these positions was received by the Branch Council with deep regret and with great appreciation of his long and devoted service. Dr. S. Gibson and Dr. A. W. Shugg were appointed to represent the Branch on the Federal Council for the present year.

FINANCIAL STATEMENT.

Dr. J. S. Reid, the Honorary Treasurer, read the financial statement for the year ended December 31, 1934. The statement, which was adopted, is published herewith.

THE BRITISH MEDICAL ASSOCIATION (TASMANIAN BRANCH).

Statement of Receipts and Payments for Year ended December 31, 1933.

RECEIPTS.

	£	s.	d.
To Balance at January 1, 1933	169	16	10
„ Members' Fees	393	18	11
„ Interest on Fixed Deposit	13	3	3
„ Refund of Vic. W. Com.	0	2	2
„ Australasian Medical Publishing Company, Limited—Interest on Debentures	18	1	11
„ Australasian Medical Publishing Company, Limited—Refund of New South Wales State Tax	0	16	11
	£596	0	0
January 1, 1934—To Balance	£308	9	11

PAYMENTS.

	£	s.	d.
By Australasian Medical Publishing Company, Limited (THE MEDICAL JOURNAL OF AUSTRALIA for 1932 and part 1931)	87	10	0
„ Remittance to London held on Fixed Deposit, E. S. and A. Bank	94	19	9
„ Federal Council Levy on Members	7	0	0
„ Hirage on Films, Articles on Cancer, and Typist's Salary	10	10	11
„ Auditors' Fees	2	2	0
„ Rent	4	0	6
„ Rules of Branch (Printing)	18	0	5
„ C.A. Fee	0	10	0
„ Exchange	0	12	8
„ Secretary's Honorarium	30	0	0
„ Petty Cash and Assistant's Salary	22	15	4
„ Duplicating, Printing and Stationery	9	8	6
„ Balance	308	9	11
	£596	0	0

Statement of Assets.

	£	s.	d.
Furniture at University	20	0	0
Cash in Hand	111	10	4
Cash at E. S. and A. Bank	196	19	7
Fixed Deposit, E. S. and A. Bank	400	0	0
Deposit with Hobart Savings Bank	150	0	0
Australasian Medical Publishing Company, Limited	95	0	0
	£973	9	11
E. S. and A. Bank, Fixed Deposit held on Account London Office	£288	13	7

We have to report that we have audited the Books and Vouchers of the British Medical Association (Tasmanian Branch) for the year ended December 31, 1933, and the above is a correct statement of Receipts and Payments for that period as shown by the books.

(*Signed*) ADAMS AND BENNETTO, Auditors,
Chartered Accountants (Aust.).

ELECTION OF OFFICE-BEARERS.

The following members were elected as office-bearers for the ensuing year:

President-Elect: Dr. G. E. Clemons.
Vice-President: Dr. E. Brettingham-Moore.
Honorary Secretary: Dr. J. H. B. Walch.
Honorary Treasurer: Dr. J. S. Reid.
Members of Council: Dr. F. W. Fay, Dr. T. H. Goddard, Dr. J. Stoddart Barr.

Messrs. Adams and Bennetto were reappointed auditors.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

Dr. D. H. E. Lines, President of the Fourth Session of the Australasian Medical Congress (British Medical Association), spoke briefly of the general work of the Congress. Dr. F. W. Fay, Honorary Secretary of the Congress Entertainment Committee, described the activities of his committee; and Dr. S. G. Gibson, Honorary Treasurer of Congress, read the Congress financial statement.

THE NORTHERN DIVISION OF THE BRANCH.

Dr. J. L. Grove read a report of the activities of the Northern Division of the Branch. This report is published below.

ALTERATIONS OF BRANCH RULES.

Alterations to the rules of the Branch were adopted as follows:

1. Rules 4, 15, 18, and 30 of the rules of the Branch were amended by substituting the words "Federal Council" for the words "Federal Committee".

2. Sections (*a*), (*b*), and (*c*) of Rule 15 were annulled and the following three sections were substituted for them:

(*a*) The Branch Council shall, in the month of December in each year, elect two members of the Association, willing to act, to represent the Branch on the Federal Council, and shall forward the Secretary of the Federal Council a notification of the result of such election so that the same shall be received by the Secretary not later than the thirtieth day of January next succeeding the date of election.

(*b*) In the event of the membership of any member representing the Association as a member of the Federal Council ceasing otherwise than by expiration of his term of membership, the Council shall elect another member in the place of the member whose membership has so ceased and shall forthwith notify the Federal Council of the result of such election. Such election shall be held with all expedition and at the latest within three calendar months of its receiving notice from the Federal Council of such vacancy, or of the Council having notified the Federal Council that a member representing the Association has ceased to be a member.

(*c*) In the event of a representative of the Branch being unable to attend any meeting of the Federal Council he shall notify the Branch Council at the earliest opportunity, and the Branch Council may nominate another member of the Association, who may or may not be a member of the Federal Council, to act as a substitute; or, if the Branch Council does not nominate a substitute, the member who shall be unable to attend may appoint another member on the Federal Council to be his proxy for such meeting.

THE ANTI-CANCER CAMPAIGN.

The President and Dr. J. L. Grove gave an account of the work of the Northern Tasmanian Anti-Cancer Cam-

paign Committee. Attempts had been made, by wireless talks and articles in the Press, to educate the public in regard to the urgent need for early diagnosis and treatment of cancer. A large sum of money had been collected, but about £1,600 was still required before a deep therapy X ray plant could be purchased and installed. It had been originally intended that a separate Southern Tasmanian Anti-Cancer Campaign Committee should be formed. But it was now proposed that the Northern Committee should be disbanded and a new committee to represent the whole State should be appointed in its stead. Members approved of this proposal and instructed the incoming Council to take what steps they could to assist in carrying it out.

INDUCTION OF PRESIDENT.

Dr. W. K. McIntyre then inducted Dr. W. E. L. Crowther as President of the Branch for the ensuing twelve months.

PRESIDENT'S ADDRESS.

Dr. W. K. McIntyre read his President's address (see page 579) and the meeting terminated.

ANNUAL REPORT OF THE NORTHERN DIVISION OF THE TASMANIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

The tenth annual meeting of the Northern Division of the Tasmanian Branch of the British Medical Association was held at the Public Hospital, Launceston, on March 7, 1934.

Office-Bearers for 1933.

The following were office-bearers for the year 1933:

Chairman: Dr. C. Craig.
Vice-Chairman: Dr. J. Ramsay.
Honorary Treasurer: Dr. C. Thompson.
Honorary Auditor: Dr. G. Harris.
Honorary Secretary: Dr. G. M. W. Clemons.
Executive Committee: Dr. G. E. Clemons, Dr. W. K. McIntyre, and Dr. A. Pryde.

Monthly Meetings.

Nine (9) meetings were held, at which the average attendance was 11. At each of these meetings clinical material was discussed and papers presented.

Post-Graduate Course.

The annual post-graduate course was held from October 28 to October 31, 1933. Dr. Konrad Hiller, Honorary Physician to the Melbourne Hospital, and Dr. A. C. Colville, Honorary Surgeon to the Alfred Hospital, Children's Department, gave a series of lectures and demonstrations at the Launceston Public Hospital. Dr. Hiller's subjects were: "The Treatment of Rheumatoid Arthritis" and "The Diagnosis and Treatment of Coronary Thrombosis". In addition, he demonstrated cases in the ward. Dr. Colville lectured on "Some Common Affections of the Hip Joint in Childhood" and "Some Points in the Diagnosis and Treatment of Intracranial Tumours in Childhood", and demonstrated the method of reduction of a case of congenital dislocation of the hip joint.

The Division expresses its indebtedness to these gentlemen and its keen appreciation of the time and trouble that they necessarily incurred.

Anti-Cancer Campaign.

The money for the equipment and buildings is now in hand. Tenders for the plant are being considered and it is hoped that the plant will be installed by the middle of the year.

Library.

The library has now been catalogued and improved by the addition of a number of new journals. It is hoped to increase further the number of journals during the present year if funds permit.

Australasian Medical Congress (British Medical Association).

The Australasian Medical Congress (British Medical Association) was held in Hobart from January 15 to 21, 1934.

The following Northern members were office-bearers:

Executive Committee: Dr. W. K. McIntyre.
Vice-Presidents: Dr. J. A. Newell (Medicine), Dr. G. H. Hogg (Ophthalmology), Dr. G. E. Clemons (Paediatrics), Dr. J. Ramsay (Pathology, Bacteriology and Cancer Research).
Secretaries: Dr. R. M. W. Webster (Naval and Military), Dr. J. L. Grove (Pathology, Bacteriology and Cancer Research), Dr. C. Craig (Surgery), Dr. G. M. W. Clemons (Medicine), Dr. W. P. Holman (X Ray and Electrical Therapy).

The Division realizes the tremendous amount of work done by the Southern office-bearers, particularly the Joint Honorary Secretaries, Dr. H. N. Butler and Dr. W. L. Crowther. The Division wishes to record its appreciation of this work and offers its congratulations on the very successful running of Congress.

Office-Bearers for 1934.

Chairman: Dr. C. Thompson.
Vice-Chairman: Dr. G. E. Clemons.
Honorary Treasurer: Dr. W. R. Holman.
Honorary Auditor: Dr. G. Harris.
Honorary Secretary: Dr. G. M. W. Clemons.
Executive Committee: Dr. C. Craig, Dr. W. K. McIntyre and Dr. J. Ramsay.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Farmer, Frank Ward, M.B., B.S., 1932 (Univ. Melbourne), Base Hospital, Ballarat.

Post-Graduate Work.

ANNUAL REFRESHER COURSE IN BRISBANE.

THE Queensland Post-Graduate Committee has issued the following programme for the annual post-graduate and refresher course that will be held in Brisbane from May 24 to June 1, 1934.

Thursday, May 24, 1934.

9 a.m. to 10.30 a.m.—Dr. L. M. McKillop: Demonstration, operative treatment of malignancy, Mater Misericordiæ Public Hospital.
11 a.m. to 12.30 p.m.—Dr. J. C. Hemsley: Demonstration, pelvic repair under local anæsthesia, Mater Misericordiæ Public Hospital.
2.15 p.m. to 4 p.m.—Dr. W. Crosse: Demonstration, the diagnosis of sinus infection, Brisbane Hospital, Out-Patients' Department.
4.30 p.m. to 5.30 p.m.—Dr. W. L. Gibson: Demonstration, common eye conditions, Brisbane Hospital, Out-Patients' Department.
8.30 p.m.—Dance, Lennon's Hotel.

Friday, May 25, 1934.

9 a.m. to 10.30 a.m.—Dr. Eustace Russell: Demonstration, ward cases, Brisbane Hospital.
11 a.m. to 12.30 p.m.—Dr. V. McDowall: Demonstration, Radium Clinic, Brisbane Hospital.
2 p.m. to 3.30 p.m.—Dr. Alex Murphy: Demonstration, ward cases, Brisbane Hospital.

4.30 p.m. to 5.30 p.m.—Professor Marshall Allan: Lecture, "Recent Advances in Endocrine Control of Female Reproductive System", Geological Theatre, University.
8.15 p.m.—Council Meeting, British Medical Association Building, Adelaide Street.

Saturday, May 26, 1934.

9 a.m. to 11 a.m.—Dr. N. Sutton and Dr. A. Lee: Demonstration, minor operative surgery under local anæsthesia, Brisbane Hospital.

Monday, May 28, 1934.

9.30 a.m.—Dr. G. Kaye: Lecture-demonstration, "Modern Apparatus and Methods used in Anæsthesia", Brisbane Hospital.
11 a.m. to 12.30 p.m.—Dr. E. S. Meyers: Demonstration, operative surgery, Brisbane Hospital.
2.30 p.m. to 3.30 p.m.—Dr. Ellis Murphy: Demonstration, ward cases, Mater Misericordiæ Public Hospital.
4 p.m. to 4.30 p.m.—Dr. Flora Innes: Lecture, "Modern Control of Leprosy", Mater Misericordiæ Public Hospital.
8.15 p.m.—Mr. W. A. Hailes: Lecture, "Early Diagnosis and Treatment of Intestinal Obstruction", Geological Theatre, University.

Tuesday, May 29, 1934.

9.30 a.m.—Dr. G. Kaye: Demonstration (Group A), anæsthetics, Mater Misericordiæ Public Hospital.
10.30 a.m. to 12.30 p.m.—Short illustrated lectures, Geological Theatre, University:

Dr. S. F. McDonald, "Pyloric Stenosis".
Dr. Elliott Smith, "Placenta Prævia".
Dr. K. B. Fraser, "Injuries in the Region of the Elbow Joint in Children".
Dr. A. V. Meehan, "Periarticular Adhesions of the Shoulder Joint".

2 p.m. to 3.30 p.m.—Dr. J. Power: Demonstration, cystoscopy, Brisbane Hospital.
4 p.m.—Mr. W. A. Hailes: Lecture-demonstration, "Management of Retention of Urine", Brisbane Hospital.
8.15 p.m.—Dr. G. Kaye: Lecture, "The Choice of an Anæsthetic", Geological Theatre, University.

Wednesday, May 30, 1934.

9 a.m.—Dr. G. Kaye: Demonstration (Group B), anæsthetics, Mater Misericordiæ Public Hospital.
9.30 a.m. to 10.30 a.m.—Dr. P. A. Earnshaw: Demonstration, ward cases, Mater Misericordiæ Children's Hospital.
11 a.m.—Dr. C. Bickerton Blackburn: Demonstration, the differential diagnosis of enlargement of the spleen, Mater Misericordiæ Public Hospital.
Afternoon—Free.

Thursday, May 31, 1934.

9 a.m.—Dr. G. Kaye: Demonstration (Group C), anæsthetics, Brisbane Hospital.
9.30 a.m. to 10.30 a.m.—Dr. B. Barrack: Demonstration, common skin conditions, Brisbane.
11 a.m. to 12.30 p.m.—Dr. H. McLelland: Demonstration, chronic cervicitis, Brisbane Hospital.
2 p.m. to 3.30 p.m.—Dr. C. Wassell: Demonstration, methods of tonsillectomy, Brisbane Hospital.
4 p.m.—Dr. C. Bickerton Blackburn: Demonstration, the differential diagnosis of enlargement of the lymphatic glands, Brisbane Hospital.
7.30 p.m.—British Medical Association Dinner, Rowe's Café.

Friday, June 1, 1934.

9 a.m.—Dr. G. Kaye: Demonstration (Group D), anæsthetics, Brisbane Hospital.
9.30 a.m. to 10.30 a.m.—Dr. J. V. Duhig: Demonstration, pre-transfusion blood typing and the organization of a donor service, Geological Theatre, University.
11 a.m. to 12.30 p.m.—Dr. B. L. W. Clarke: Demonstration (X ray), common bone conditions, Geological Theatre, University.
2.15 p.m.—Mr. W. A. Hailes: Demonstration, the anatomy, diagnosis and treatment of hepatic, subphrenic and pleural collections, Geological Theatre, University.
4.30 p.m.—Dr. G. Kaye: Lecture, "The Uses and Abuses of Carbon Dioxide", Geological Theatre, University.
8.15 p.m.—Dr. C. Bickerton Blackburn: Bancroft Oration, "The Nervous Factor in Disorders of the Heart", Geological Theatre, University.

LECTURES IN SYDNEY.

THE New South Wales Permanent Post-Graduate Committee announces that, concurrently with the refresher course for 1934, two lectures will be given by Professor F. Wood Jones, of the University of Melbourne. The lectures will be given in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, as follows:

Tuesday, May 22, 1934: "A Study of the Spinal Column."
Thursday, May 24, 1934: "Some Findings of Experimental Embryology."

The lectures will start at 8.15 p.m. Those wishing to attend the lectures should without delay notify the Honorary Secretary, New South Wales Permanent Post-Graduate Committee, 225, Macquarie Street, Sydney, enclosing a cheque for one guinea, to which exchange should be added.

COURSE OF INSTRUCTION IN MELBOURNE FOR THE PRIMARY FELLOWSHIP EXAMINATION OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND.

THE Melbourne Permanent Post-Graduate Committee announces that a course of instruction for the Primary Fellowship Examination of the Royal College of Surgeons of England will begin early in August, 1934, and will continue until shortly before the examination on November 29, 1934. The lecturers will be:

Anatomy: Professor F. Wood Jones.
Physiology: Professor W. A. Osborne, Dr. C. H. Kellaway.

There will be two tutorial classes a week in each subject. The time-table will be arranged by the lecturers after consultation with the class. The fee for the complete course will be twenty guineas. Candidates who require dissecting parts must, in addition, pay the usual University fees for these to the Registrar of the University.

Prospective candidates must notify the Secretary of the Melbourne Permanent Post-Graduate Committee, 61, Collins Street, Melbourne, on or before July 9, of their intention to attend this course of instruction, and must enclose a cheque for the fee with this notification.

Candidates are reminded that it is necessary to enter for the examination on or before August 1, 1934.

Entrance forms are obtainable from the Censor-in-Chief of the Royal Australasian College of Surgeons, 6, Collins Street, Melbourne.

Obituary.

HAROLD SELWYN CAPPER.

DR. J. L. T. ISBISTER has written the following appreciation of the late Dr. Harold Selwyn Capper.

Dr. Harold Selwyn Capper, of North Sydney, who died suddenly on March 13, was very well known and much respected by a very large circle of friends and patients.

He was born in 1873 and was educated at the Sydney Grammar School. He entered the medical course at University College, London, where he took the Fellowes Clinical Medal in 1898 and a gold medal for first place in medicine. He took the L.R.C.P. and M.R.C.S., and in 1901 the M.D. degree of London.

He returned to his native land in 1902 and practised in the northern suburbs of Sydney up to within a fortnight of his death. For many years he was on the staff of the Royal North Shore Hospital, from which he retired a few years ago.

He was universally liked and respected by all classes of the community. His kindness of heart was proverbial, and none appealed to him in vain. To him it was immaterial whether his poorer patients could pay him for his services or not. He was a student and a great reader, of a very retiring disposition, and always most careful to avoid publicity in any form whatever. His passing will be deeply mourned.

JOHN JOSEPH HANLY.

DR. JOHN JOSEPH HANLY who died at Croydon, Victoria, on March 18, 1934, was born at Cloughjordan, County Tipperary, Ireland, on March 23, 1859. He began his university career in the Faculty of Arts of the Royal University of Ireland. He obtained a scholarship at each examination in his arts course. He graduated as Bachelor of Arts with first class honours in biological science and subsequently gained the Senior Scholarship in Natural History at Galway Queen's College. He became Master of Arts in 1881. Hanly then turned to medicine; he studied first of all in Ireland and then in Edinburgh, gaining the diploma of licentiate of the Royal College of Physicians and of the Royal College of Surgeons of Edinburgh in 1884. He held several resident appointments in Great Britain and came to Melbourne in 1909. For a while he practised in Queensland. He practised at Croydon, Victoria, until the time of his death.

Correspondence.

ACUTE BILATERAL EXTERNAL OTITIS DUE TO BACILLUS PYOCYANEUS.

SIR: On page 17 of your issue of January 6, 1934, Dr. Bettington gives a typical history and an excellent description of what the tropical practitioner would term a "neglected case of tropical ear". The patient, a male, just landed from a three months' Eastern tour, including Java and Malaya, was seen by him in the second week of the illness, on November 7, 1932, over a year before publication of the report, which is titled "Acute Bilateral External Otitis due to Bacillus Pyocyaneus", after which statement there should have appeared a query in brackets surely, when the report concludes with the sentence: "I was unable to gain any idea of how the infection arose in this case, but report the case as showing the extreme virulence of an organism in the tropics which appears to be much more benign in colder climates."

It appears that the author had his doubts and withheld the publication, hoping for similar cases to come to his notice. Any tropical practitioner could assure him that *Bacillus pyocyaneus* is no more virulent in the tropics than elsewhere. Furthermore, it is not met with in the tropical ear in the first week. Seeing that the early stages of this ear condition constitute more than 90% of our aural cases, it will be realised how important the condition is and how it is known and dreaded by the European residents. In New Guinea the sufferer will explain that he has the coral insect in his ear, or coral poisoning; in Malaya, I believe, the condition is known as "Singapore ear".

The first lesion is a minute vesicle, on a mildly inflamed base, with œdema of the surrounding skin at or outside the external meatus and usually below it, but inside the pinna. One sudden intolerable itch and the vesicle is ruptured by the finger and there ensues pain and exquisite tenderness. However, the fine *peau d'orange* œdema remains and is a warning to the practised eye not to insert a speculum.

You are dealing with a form of moniliasis and the causative fungoid organisms must be treated with respect when they are near such a warm, dark, moist cavern.

First, turn to the unaffected ear and insert a sterile speculum; paint the external auditory canal with 2% mercurochrome solution in water; warn the patient not to touch this ear with a finger under any consideration. Now paint the affected area of bad ear; allow to dry. Next, with a capillary pipette inserted well into the canal, and avoiding the affected area, swill a few minims of the mercurochrome solution down and up the pipette, using the Wright capillary tube as you would when inoculating fluid media in bacteriological technique.

Some practitioners pin their faith on 1% *hydrargyri perchloridum*, used similarly.

Make no attempt to syringe out the ear. Lastly, fill the affected ear with an efficient fungicide, which will also relieve the pain and the itch. In my practice I use "Mycozol" (Parke, Davis), rubbed up to a thin cream with "Chloretone Inhalant" and dispensed in a Stearn's dropper.

Instruct the patient to fill the ear three times daily and insert a pledget of wool inside the pinna during continuance of symptoms. Warn the patient against using this dropper for the good ear, unless it definitely becomes affected. Warn him also that the bad ear will not improve if a finger or a match is even occasionally inserted and the ear is given a satisfactory scratch. Advise use of the dropper two to three times a week for two months after apparent cure.

As a first-aid dressing, "Dentalone", with its chlorbutal and essential oils, gives the patient a welcome relief. This treatment will abort the threatening external otitis.

Should the patient be seen in the next stage, when the skin of the canal itself is involved, the meatal margin will be found weeping and the canal walls approaching or in contact. The pain will be neuralgic along both upper and lower jaws, will shoot to the top of the head and to the neck, and movement of the jaw will initiate an attack. You will think of a furuncle in the external auditory meatus and might act accordingly. I have been informed that in this stage the dammed back secretion can erode the tympanic membrane and cause a perforation, but I have not seen an example of this happening. I have seen the ears, after the incision down to bone seeking pus in the suspected furuncle cases, though.

In this, the second stage, the paraffin base drops recommended above give a little comfort, but you will have to rely on diathermy, infra-red radiation and morphine for relief of pain.

The next stage is where you will find the swelling extending over the mastoid process, down the neck and across the cheek—the pinna standing away from the head and a profuse discharge from the ear. The skin of the pinna will be eczematoid in appearance, the jaw clenched and the pains previously described exaggerated. You will relieve the patient considerably with intravenous calcium and parathormone, in addition to the electrotherapy above mentioned, but he will be miserable for a week. Try the incisions, vaccine treatment, milk injections or protein shock and you will have a nervous wreck only fit for a vacation after one or two months' intensive treatment. I fancy "Omnadin" is useful in the neglected cases, but it is difficult to be certain. Intense allergic phenomena sometimes supervene; hence the calcium and parathyreoid in prophylaxis and treatment.

You are now conversant with one phase of a skin disease, so follow the established routine in skin rash cases and examine carefully the skin of the whole body. You will invariably find a lesion which antedates the ear infection. Quite possibly it is a dhobie itch, picked up in the tropics, or cracked toes, which occasionally give trouble and which you will be told is "only tinea". Look carefully and you will see remnants of small vesicles; then you will get a history of itching skin rashes, for example, prickly heat, which is definitely known to be caused by a monilia.

The patient has probably been swimming for long periods in the warm tropical sea. The itch of the rash increased

considerably after drying in the sun, and the finger nails were used to some purpose; then the toes were carefully dried and the spaces between got the final touch with the little finger. Next, this finger goes into the ear with its sodden epidermis, where it is twisted round and round, the head inclined to that side, and perhaps one or two jumps given to get the water out of the ear. Then it is not a case of "on the evening of the third day the discharge was first noticed", but "on the next day the itch was felt". Advise against drying between the toes and against drying the ears at all. The tropical resident, once bitten, recognizes the itch—the coral insect before mentioned. In Australia, you will call it eczema and you will wonder why salicylic acid, silver nitrate, zinc salts, and such like fungicides are recommended and have been used empirically for generations for "catarrh" of the skin. In this connexion it is interesting to recall that Sir Aldo Castellani stated that thirty years ago the science of bacteriology was in the state that mycology or the study of the pathogenic fungi is at the present time. In Singapore, I understand that a tinea has been isolated as the primal causative organism of the Singapore ear. In my experience a monilia, probably closely related to, if not identical with, *Monilia pinoyi*, is involved. It is of interest that a colleague had ear trouble on his own person with *Bacillus pyocyaneus* for five years; after failing with vaccines and the usual treatment, one treatment with eusol was sufficient to cure it. So much for *Bacillus pyocyaneus*.

Some months back Dr. Ivan Connor showed monilia in perionychia cases and pointed out some of the difficulties involved in the isolation of these fungi. That paper has created no apparent stir to date.

Even in the tropics it is only slowly that the wealth of clinical material associated with the mycoses, dermal and visceral, forces the newcomer, whose scientific vision is obscured by preconceived, or rather ingrained, notions of the relative importance of staphylococci, streptococci, bacilli, spirochætes, viri *et cetera*, to the exclusion of the humble but important fungi, to realize that there is still a vast field of organisms with which to become conversant.

Yours, etc.,

ALLAN SPENCE, M.B., B.S. (Melb.).

Rabaul,
April 10, 1934.

THE TREATMENT OF UNUNITED FRACTURES.

SIR: The article by Dr. N. D. Royle in your journal of the 24th ultimo calls for some friendly criticism before it can finally be accepted as an improvement in the treatment of ununited fractures.

From my point of view, the following points arise: It would have been more appropriate if the title of the article had been "A Method of Treatment of Ununited Fractures". To the problems in treatment of these fractures I would add: (iii) to remove, where possible, any cause delaying union or producing non-union. This would then call for a slight modification of the subsequent passages in the article.

Regarding the ultimate fate of the beef bone screws, it requires many years for their absorption to take place. Consequently they must be looked upon as foreign bodies, and therefore their use is to be deprecated.

For practical purposes one accepts the behaviour of the graft, as stated in the article, although it would be more correct to say, in the present state of our knowledge, that "the graft is replaced by new bone".

The provision of the exoskeleton is most important, but I fail to see why "Cellona" bandages are thus extolled, as any well made plaster of Paris bandages, using a good grade of plaster, make an excellent splint for this purpose, are much more economical than celluloid (from the patient's viewpoint), and can be applied by the surgeon himself, thus obviating the need of a Mr. Bennett, which fact renders the method more generally applicable, and thus more likely to be accepted, than the one set out.

The unpadded plaster cast is one of the causes of delayed union of fractured tibiæ, and therefore its universal use is not advocated. Time and trial will discover what advantages this method may have.

Yours, etc.,

A. R. HAMILTON.

British Medical Association House,
135, Macquarie Street,
Sydney,
April 5, 1934.

TUBERCULIN.

SIR: I wish to join with Dr. W. Bruce Fry in his remarks (March 24, 1934) *re* the omission of any reference to the use of tuberculin in diagnosis and treatment from the syllabus for the post-graduate course on tuberculosis.

I am only a recent convert from the present-day teaching that tuberculin is not of great importance in the diagnosis *et cetera* of tuberculous troubles. My older conception of tuberculin had to be modified when I began to use it in the diagnosis and treatment of many forms of chronic sickness.

I feel convinced that many patients received great benefit when given tuberculin injections, either alone or as an adjuvant to other treatment for their special conditions.

Injections were only given to those that proved markedly sensitive to intracutaneous injections of tuberculin. Quite a few cases proved to have tuberculous lesions when diagnosed by further tuberculin injections, although they had proved negative to X ray and sputum analysis, and in these cases especially the benefit was evident—cases such as chronic bronchitis, pleurisy and so-called nervous breakdown.

I am optimistic for the future of tuberculin in diagnosis and treatment of chronic tuberculous troubles, either alone or when associated with other chronic infections.

Yours, etc.,

K. K. SPENCE, M.B., Ch.M.

51, Sophia Street,
Seven Ways,
Bondi Beach,
New South Wales.
Undated.

THE TREATMENT OF ACUTE ENTERITIS IN INFANTS.

SIR: In the very excellent account of the treatment of infantile gastro-enteritis by Dr. Helen Mayo in your issue of April 21, it is rather striking that there is no mention of the raw apple treatment of this condition. Originally described in Germany by Moro, it has attracted comparatively little notice, but those who have tried it find it most impressive in its effects. Its use requires a considerable degree of courage at first, and some users are disappointed, but personally I would try it on any occasion first, even in the severe toxic cases.

The best account I have seen in English is that of T. L. Birnberg in the *American Journal of Diseases of Children*, January, 1933 (Volume XLV, Number 1, page 18). A summary, almost a *verbatim* reprint, is published in the 1933 "Year Book of Pediatrics" (Year Book Publishers, Incorporated, Chicago).

Possibly Dr. Mayo has had unsatisfactory results with this treatment; others consider it the greatest advance in the treatment of infantile digestive disturbances since Marriott introduced lactic acid milk.

Yours, etc.,

S. F. MCDONALD, M.D., M.R.C.P.

"Craigston",
217, Wickham Terrace,
Brisbane,
April 24, 1934.

Books Received.

ANATOMY OF ANIMAL TYPES FOR STUDENTS OF ZOOLOGY, by E. A. Briggs, D.Sc., 1934. Australia: Angus and Robertson. Demy 8vo., pp. 270. Price: 10s. 6d. net.

VOLUMETRIC ANALYSIS, by H. P. Starck, M.A.; 1934. London: Baillière, Tindall and Cox. Demy 8vo., pp. 236, with illustrations. Price: 7s. 6d. net.

A TEXTBOOK OF THE PRACTICE OF MEDICINE, by various authors, edited by F. W. Price, M.D., F.R.S.; Fourth Edition; 1933. London: Humphrey Milford (Oxford University Press); Australia: Angus and Robertson. Royal 8vo., pp. 2040. Price: 56s. net.

CLINICAL INVESTIGATION OF CARDIOVASCULAR FUNCTION, by V. Pachon and R. Fabre, translated by J. F. Halls Daly, M.A., M.D., M.R.C.P.; 1934. London: Kegan Paul, Trench, Trubner and Company, Limited. Demy 8vo., pp. 263, with illustrations. Price: 15s. net.

DIATHERMY IN GENERAL PRACTICE, by E. P. Dark, M.B., Ch.M., with chapters on Diathermy in Gynæcology, with special reference to Major Surgery, by F. A. Maguire, M.D., Ch.M., F.R.C.S., F.R.A.C.S., and Diathermy in the treatment of Mental Disorders, by G. P. U. Prior, L.R.C.P., M.R.C.S.; Second Edition; 1934. Australia: Angus and Robertson, Limited. Demy 8vo., pp. 231, with illustrations. Price: 17s. 6d. net.

NEUROANATOMY: A GUIDE FOR THE STUDY OF THE FORM AND INTERNAL STRUCTURE OF THE BRAIN AND SPINAL CORD, by J. H. Globus, B.S., M.D.; Sixth Edition, revised and enlarged; 1934. London: Baillière, Tindall and Cox. Demy 4to., pp. 266, with illustrations. Price: 16s. net.

THE ORIGIN OF CANCER, by J. P. Lockhart-Mummery, M.A., M.B., B.C., F.R.C.S.; 1934. London: J. and A. Churchill. Demy 8vo., pp. 167, with illustrations. Price: 10s. 6d. net.

Diary for the Month.

May 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
May 8.—Tasmanian Branch, B.M.A.: Branch.
May 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
May 11.—Queensland Branch, B.M.A.: Council.
May 15.—New South Wales Branch, B.M.A.: Ethics Committee.
May 15.—Tasmanian Branch, B.M.A.: Council.
May 16.—Western Australian Branch, B.M.A.: Branch.
May 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
May 23.—Victorian Branch, B.M.A.: Council.
May 24.—New South Wales Branch, B.M.A.: Clinical Meeting.
May 25.—Queensland Branch, B.M.A.: Council.
May 31.—South Australian Branch, B.M.A.: Branch.
May 31.—New South Wales Branch, B.M.A.: Branch.
June 1.—Queensland Branch, B.M.A.: Branch: Bancroft Memorial Lecture.
June 5.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments.

Dr. V. Lynch (B.M.A.) has been appointed Medical Inspector of Seamen for the Port of Gladstone, Queensland, pursuant to the provisions of the *Navigation Act*, 1912-1926.

* * *

Dr. E. Sandford Jackson (B.M.A.) has been appointed Medical Inspector of Seamen for the Port of Townsville, Queensland, pursuant to the provisions of the *Navigation Act*, 1912-1926.

* * *

Dr. W. H. Wallace has been appointed Government Medical Officer at Long Jetty (Tuggerah Lakes), New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

Royal North Shore Hospital of Sydney, New South Wales: Temporary Honorary Assistant Surgeon.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
South Australian: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

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